

Utah State University

**DigitalCommons@USU**

---

All Graduate Theses and Dissertations

Graduate Studies

---

5-2010

## Depression Detection in Hospitalized Cardiac Patients

Martine S. Geddes

*Utah State University*

Follow this and additional works at: <https://digitalcommons.usu.edu/etd>



Part of the [Public Health Education and Promotion Commons](#)

---

### Recommended Citation

Geddes, Martine S., "Depression Detection in Hospitalized Cardiac Patients" (2010). *All Graduate Theses and Dissertations*. 630.

<https://digitalcommons.usu.edu/etd/630>

This Thesis is brought to you for free and open access by the Graduate Studies at DigitalCommons@USU. It has been accepted for inclusion in All Graduate Theses and Dissertations by an authorized administrator of DigitalCommons@USU. For more information, please contact [digitalcommons@usu.edu](mailto:digitalcommons@usu.edu).



DEPRESSION DETECTION IN HOSPITALIZED  
CARDIAC PATIENTS

by

Martine S. Geddes

A thesis submitted in partial fulfillment  
of the requirements for the degree

of

MASTER OF SCIENCE

in

Health and Human Movement

Approved:

---

Phillip J. Waite, Ph.D  
Major Professor

---

Julie Gast, Ph.D  
Committee Member

---

Kim Openshaw, Ph.D  
Committee Member

---

Byron Burnham, Ph.D  
Dean of Graduate Studies

UTAH STATE UNIVERSITY  
Logan, Utah

2010

Copyright © Martine S. Geddes 2010

All Rights Reserved

## ABSTRACT

Depression Detection in Hospitalized  
Cardiac Patients

by

Martine S. Geddes, Master of Science  
Utah State University, 2010Major Professor: Phillip J. Waite, Ph.D.  
Department: Health, Physical Education, and Recreation

Depression has been shown to increase an individual's risk for heart disease. Despite this finding, physicians are not identifying depression in their hospitalized cardiac patients. This study looked at hospitalized cardiac patients and determined whether their physicians were identifying depression in those that scored  $\geq 5$  on the PHQ-9 depression inventory. Methods included assessing patient depression during their stay at an intensive care unit using the PHQ-9. Those patients scoring  $\geq 5$  were determined as depressed. Chart audits were performed after the patient discharged from the hospital to discover whether physicians were identifying these patients as depressed. The results showed that out of 111 surveys, 83 had a score of  $\geq 5$ , meaning that 74.7% of hospitalized cardiac patients have some type of depression while in the hospital, ranging from mild, moderate, or severe. Of those 83 patients, only 9 or 10% were identified as depressed by their physician during their stay at the hospital. Conclusions suggest that although depression appears to be prevalent in the hospitalized cardiac patient, physician detection of such is very low.

(114 pages)

## ACKNOWLEDGMENTS

The completion of this project was made possible by so many individuals with special thanks to my committee chair, Dr. Phillip J. Waite, and committee members, Dr. Julie Gast and Dr. Kim Openshaw. And in memory of Dr. Mary Doty, who began as one of my committee members and lent her very gracious criticism and support prior to her death.

A special thank you must be given to Phil Waite and Julie Gast. Julie always encouraged me to tackle each portion of my thesis in “chunks.” No better advice was ever given. Phil taught me about persistence and perseverance during the course of my studies. His guidance on chapters four and five was immeasurable.

To my husband and my children, thank you. Your support and sacrifice of your wife and mother was not easy, but was much appreciated. Education is so important in this world, and I am grateful to Intermountain Healthcare for providing me the opportunity to grow and learn in ways that I never could have without their help. To my children, always strive for more education. This will help you go where you want to in life.

Martine S. Geddes

## CONTENTS

	Page
ABSTRACT.....	iii
ACKNOWLEDGMENTS .....	iv
LIST OF TABLES.....	viii
CHAPTER	
I. INTRODUCTION .....	1
Background of the Problem .....	1
Problem Statement .....	3
Purpose of the Study .....	4
Research Questions .....	4
Limitations .....	4
Delimitations.....	5
Operational Definition of Terms.....	5
II. REVIEW OF THE LITERATURE.....	7
The Physiological Relationship Between Depression and Heart Disease.....	7
Increased Platelet Aggregation/Activation and Depression.....	7
C-Reactive Protein.....	9
Cortisol Levels.....	13
Risk Factors for Depression.....	14
Positive Family History of Depression.....	15
Psychological/Life Stressors.....	19
Smoking.....	24
History of CHD/MI.....	27
Social Isolation/Social Support.....	29
Type D Personality .....	34
Gender.....	39
NYHA Class .....	44
The Prevalence of Depression in the Cardiac Patient.....	48
Following Hospitalization for MI.....	48
Following Hospitalization for CABG.....	52
Following Hospitalization for CHF .....	55
The Effects of Untreated Depression.....	58

	Increased Risk of a Coronary Event .....	59
	Decreased Medication Compliance .....	61
	Decreased Feelings of Wellbeing/Quality of Life .....	61
	Physician Under Detection of Depression .....	62
III.	METHODOLOGY .....	66
	Research Design .....	66
	Sample .....	66
	Selection Criteria .....	65
	Sample Size .....	67
	Sample Characteristics .....	67
	Instrumentation .....	69
	Pilot Testing.....	71
	Data Collection .....	71
	Data Analysis.....	73
	Summary .....	74
IV.	RESULTS .....	75
	Question Number One .....	75
	Question Number Two .....	76
	Question Number Three .....	77
	Question Number Four .....	78
	Question Number Five .....	79
	Question Number Six .....	79
	Summary .....	80
V.	DISCUSSION .....	81
	Introduction .....	81
	Question Number One .....	81
	Question Number Two .....	83
	Question Number Three .....	84
	Question Number Four .....	85
	Question Number Five .....	85
	Question Number Six .....	86
	Limitations of the Study .....	88
	Implications for Health Education .....	89
	Summary .....	90
	REFERENCES .....	91

APPENDICES .....	100
Appendix A: Patient Health Questionnaire (PHQ-9).....	101
Appendix B: Informed Consent .....	105
Appendix C: Pilot Study Evaluation Form .....	108
Appendix D: Physician Letter and Consent Form .....	110
Appendix E: Letter of Approval Intermountain IRB .....	113



## LIST OF TABLES

Table	Page
1. Nomenclature and Criteria for Diagnosis of the Heart and Great Vessels.....	44
2. Demographic Characteristics of the Sample .....	68
3. Research Questions and Data Analysis Procedures .....	74
4. Patient Depression Detection .....	76
5. Depression Among Diagnostic Groups.....	77
6. Male and Female Depression .....	78
7. Marital Status and Depression .....	78
8. Variables in the Equation .....	79
9. Prior and New Cardiac Diagnosis and Depression .....	80

## CHAPTER I

### INTRODUCTION

#### **Background of the Problem**

Evidence suggests that depression promotes atherosclerosis in the cardiac patient (Glassman, 2007; Goldstein, 2006; Januzzi & Pasternak, 2002). Depression can increase chronic inflammation in the arteries, platelet abnormalities, and other side effects that can result from chronic stimulation of the sympathetic nervous system (Feinstein, Blumenfeld, Orlowski, Frishman, & Ovanessian, 2006). Depressive symptoms, whether formally diagnosed as clinical depression or not, can be strong independent predictors of mortality, worse health status, decreased medical compliance, recurrent cardiac events, and increased patient usage of health care services (Amin, Jones, Nugent, Rumsfeld, & Spertus, 2005).

Increased platelet aggregation/activation has been linked to depression (Feinstein et al., 2006; Goldstein, 2006; Januzzi & Pasternak, 2002; Lane, Carroll, & Lip, 1999). Higher levels of platelet factor IV and beta thromboglobulin have been demonstrated in patients with depression and have been found to increase thrombus formation. Depression increases the levels of c-reactive protein (CRP), which is a marker for an inflammatory process in the artery walls thought to be a predictor of coronary heart disease (CHD) (Feinstein et al., 2006; Goldstein, 2006; Januzzi & Pasternak, 2002). Research shows that depression can raise cortisol levels in the blood. High cortisol levels may increase endothelial inflammation by excess clotting. This has been found to be associated with hypertension, increased cholesterol, and glucose dysregulation (Januzzi & Pasternak, 2002; Kemp, Malhorta, Franco, Tesar, & Bronson, 2003).

Evidence shows that there may be certain risk factors that influence the development of depression for the cardiac patient. A positive family history of depression is the most prevalent

risk factor (Lesperance & Frasere-Smith, 2000). Although psychological stressors do not always indicate risk factors for depression, they can be a potential cause (Thornton, 2001). There are certain clusters of risk factors for depression that are also well-known risk factors for CHD and include: smoking, disability or poor New York Heart Association Class (NYHA) stress and high workload, and history of CHD (Lehto et al., 2000). Social isolation and poor social support have been shown to increase an individual's risk for developing depression (Kemp et al., 2003; Lesperance & Frasere-Smith, 2000; Thornton, 2001).

A newly emerging idea that is receiving a great deal of attention is called the type "D" personality. This personality type is considered a high-risk personality and is characterized by negative affectivity and social inhibition combined (Thornton, 2001). This personality type is rapidly replacing the highly regarded notion of the type "A" personality in regards to CHD. Each of these risk factors for depression will be discussed below.

The prevalence of depression among cardiac patients can range anywhere from 15-65% (Amin et al., 2005; Feinstein et al., 2006; Herrmann-Lingen, 2001; Kaptein, De Jonge, Van Den Brink, & Korf, 2006). Despite this knowledge, physicians, including cardiac physicians, are under-detecting this problem (Amin et al., 2005; Herrman-Lingen, 2001; Huffman et al., 2006; Januzzi & Pasternak, 2002). Evidence suggests that depression experienced after a myocardial infarction (MI) is not transient. At 3, 6, 9, and 12 months post-MI, research has found that patients may still meet criteria for moderate to severe depression (Frasere-Smith, 2000; Lane et al., 1999).

The effects of untreated depression can seriously complicate recovery and increases the risks of further cardiac morbidity or mortality (Carney, Freedland, Sheline, & Weiss, 1996; Kemp et al., 2003). The more severe the depression is, the greater the risk of death following a cardiac event (Kemp et al., 2003). Non-adherence to a physician's medication treatment plan is increasingly recognized and can cause adverse cardiac outcomes (Ali, Gehi, Na, & Whooley,

2007).

The American College of Cardiology (ACC) and the American Heart Association (AHA) recommend routine assessments into depressive symptoms in the cardiac patient in order to establish appropriate interventions and follow up (Amin et al., 2005; Thornton, 2001). The use of a screening procedure can greatly improve the recognition of depression (Magruder-Habib, Zung, & Fuessner, 1990). Positive screening should receive a thorough diagnostic interview and a criteria-based diagnosis (Hermann-Lingen, 2001). The detection and treatment of depression should be a critical element in the overall rehabilitation of the cardiac patient (Lehto et al., 2000).

Prior research has been conducted to examine depression among the hospitalized MI and CHF patient (Amin et al., 2005; Huffman et al., 2006; Kaptein et al., 2006). Findings suggest that subjects with significant depressive symptoms during hospitalization that increased during the post-MI year had a significantly higher rate of new cardiovascular events, which was not explained by any initial measure of cardiac impairment (Kaptein et al., 2006). To date, however, more research is needed to investigate the prevalence of under-detection of depression among patients with other cardiac diagnoses requiring hospitalization. These diagnoses would include MI, CHF, atrial fibrillation, unstable angina, acute coronary syndrome (ACS), and rule out MI with a past history of cardiac problems.

### **Problem Statement**

Since the mid-1990s, evidence has suggested that depression can aggravate the course of multiple cardiovascular conditions (Glassman, 2007; Feinstein et al., 2006). Despite the evidence, depression detection in the hospitalized cardiac patient remains low (Amin et al., 2005; Hermann-Lingen, 2001; Huffman et al., 2006; Januzzi & Pasternak, 2002). The present state of knowledge regarding depression detection in the cardiac patient for diagnoses other than MI and CHF is very limited. A better understanding of the rate of depression detection within other diagnoses is

needed in order to better address those patient's needs.

### **Purpose of the Study**

This study was carried out in an attempt to expand the research that has already been conducted on hospitalized MI and CHF patients by assessing the rate of detection of depression among hospitalized patients diagnosed with a wide spectrum of cardiac related disorders.

### **Research Questions**

The following research questions were addressed in this study:

1. Are cardiologists and/or internal medicine physicians detecting depression in their cardiac patients during a hospital admission accurately as compared to the PHQ-9?
2. Are there differences between the rates of depression in the different diagnostic groups?
3. Are there differences between the rates of depression in male and female patients?
4. Are there differences between the rates of depression in patients who are married, single, widowed, or divorced?
5. Does the age of a patient predict the risk for depression following a cardiac event?
6. Are there differences between the rates of depression in patients that had a prior cardiac history versus those with a new diagnosis?

### **Limitations**

The limitations of the study are listed below:

1. Some ( $n = 4$ ) patients were transferred to a hospital that provides a higher level of care in a very short period of time, which made it impossible for those patients to participate in the study.

2. Due to the fast-paced nature of an intensive care unit, administering the chosen depression inventory was not feasible with some patients (21 individuals); i.e., those preparing for transfer ( $n = 4$ ), patients with altered levels of consciousness ( $n = 5$ ), the inability of the researcher to administer the PHQ-9 due to discharge of the patient ( $n = 9$ ), and those that declined to fill out ( $n = 3$ ).

### **Delimitations**

The delimitations of the study are listed below:

1. The sample captured in this study is unique to this region and may not be generalizable to other populations of cardiac patients.
2. All patients were 18 years of age or older.

### **Operational Definition of Terms**

For the purpose of this study, the following terms and definitions were used.

*Coronary Heart Disease (CHD)*: Myocardial damage due to insufficient blood supply.

The disease is caused by pathological changes in the coronary arteries sufficient to interfere with adequate blood flow (Davis, 1978).

*Coronary Artery Bypass Grafting (CABG)*: A technique that involves using the patient's greater saphenous vein, as a bypass graft between the proximal aorta and the diseased coronary artery distal to an atherosclerotic lesion. The internal mammary or radial arteries can also be used (Ehrman, Gordon, Visich, & Kateyian, 2003).

*Congestive Heart Failure (CHF)*: A condition in which the heart is unable to pump enough blood to meet the needs of the body. This condition occurs because the heart becomes weakened by conditions or diseases that damage the heart muscle (Intermountain Health Care, 2005).

*Cortisol:* An adrenal cortical hormone, usually referred to as hydrocortisone. It is important due to its regulatory action in metabolism of fats, carbohydrates, sodium, potassium, and proteins (Davis, 1978).

*Depression:* According to the Patient Health Questionnaire (PHQ-9), a score of 1-4 indicates minimal depression, 5-9 indicates mild depression, 10-14 indicates moderate depression, 15-19 indicates moderately severe depression, and 20-27 indicates severe depression (see Appendix A).

*Myocardial infarction (MI):* A clinical condition caused by occlusion of a coronary artery or branches of the arteries that results in heart damage due to a part of the heart muscle being deprived of oxygen for a prolonged period of time (Ehrman et al., 2003).

*Platelets:* Platelets play an important role in blood coagulation, hemostasis, and blood thrombus formation. When a small vessel is injured, platelets adhere to each other and the edges of the injury form a plug that covers the area. The plug, or blood clot formed soon retracts and stops the loss of blood (Davis, 1978).

*New cardiac diagnosis:* The first time a patient has been diagnosed with anything that is cardiac related.

*Prior cardiac diagnosis:* A cardiac diagnosis that is not the first cardiac diagnosis for a given patient. The patient has a prior history of cardiac problems.

## CHAPTER II

### REVIEW OF THE LITERATURE

This chapter will review recent literature concerning the relationship between depression and heart disease and the detection of this problem by physicians. The literature review will examine the physiological relationship between depression and heart disease and will also include: (a) the prevalence of depression in the cardiac patient, (b) morbidity caused by depression, (c) mortality caused by depression, (d) risk factors that precipitate depression, and (e) the prevalence of physician detection.

#### **The Physiological Relationship Between Depression and Heart Disease**

In the mid-1990s, evidence appeared to indicate that depression following an MI increased the risk of morbidity and mortality (Glassman, 2007). It was discovered that depression and pain share a common neurochemical pathway in that they are both influenced by serotonin and norepinephrine (Trivedi, 2004). Depression has become substantially more prevalent in patients with CHD than is widely recognized (Januzzi & Pasternak, 2002). This section will address the most widely accepted associations between depression and CHD.

#### **Increased Platelet Aggregation/ Activation and Depression**

Increased platelet aggregation/activation has been linked to depression (Feinstein et al., 2005; Goldstein, 2006; Januzzi & Pasternak, 2002; Lane et al., 1999). Higher levels of platelet factor IV and beta thromboglobulin have been demonstrated in patients with depression and can increase thrombus formation. Increased thrombus formation can increase the risk of thromboembolic events (Goldstein, 2006; Lane et al., 1999). Serotonin has also been shown to



increase platelet cytosolic calcium which leads to exaggerated platelet response and enhanced platelet aggregation (Goldstein, 2006).

Serebruany et al. (2003) found that platelets play a key role in the progression of ACS. Clinical depression alone is also associated with platelet activation. This study set out to compare the concentrations of established biomarkers to reveal enhanced platelet activation in clinically depressed versus nondepressed patients. The subjects were enrolled in clinical trials for ACS. Two hundred and eighty-one baseline plasma samples were drawn from patients with acute MI ( $n = 41$ ), ACS ( $n = 126$ ), clinical depression and previous ACS within 6 months ( $n = 64$ ), and from normal healthy controls ( $n = 50$ ). The blood was drawn prior to application of any therapeutic strategies. Patients with ACS showed a greater degree of platelet activation than controls independently of the presence of depression. The depressed plus ACS group had higher plasma levels of all biomarkers compared with the nondepressed patients. The results reveal that clinical depression is associated with enhanced activation of platelets even above the level expected with ACS.

Musselman et al. (1996) conducted a study to investigate whether the depressed patient showed increased platelet reactivity. In vivo platelet activation was measured in 12 depressed patients and 8 normal, nondepressed control patients after overnight bed rest and also orthostatic challenge. The results showed that depressed patients demonstrated increased platelet activation at baseline as compared to the control patients. This increased susceptibility to platelet activation could be a mechanism linking depression as a significant risk factor for ischemic heart and cerebrovascular disease and/or mortality after MI.

Lederbogen et al. (2001) conducted a study that looked at platelet aggregability in patients with major depression both prior to and after 5 weeks of antidepressant therapy as well as in healthy control subjects. The participants consisted of depressed patients ( $n = 22$ ) and healthy control subjects ( $n = 24$ ). In this study, washed and rediluted platelets were stimulated.

Depression was associated with a higher aggregability after stimulation. Following 5 weeks of antidepressant therapy, aggregability was somewhat less exaggerated, but the effect unfortunately did not reach statistical significance.

Although contrasting information is available, the research tends to sway in the direction of increased platelet aggregability when depression is present (Lederbogen et al., 2001; Musselman et al., 1996; Serebruany et al., 2003). Even without achieving statistical significance in one study, it was still demonstrated that platelet aggregation had decreased following antidepressant therapy (Lederbogen et al., 2000).

### **C-Reactive Protein (CRP)**

Depression increases the levels of CRP, which is a marker for an inflammatory process in the artery walls thought to be a predictor of CHD (Feinstein et al., 2005; Goldstein, 2006; Januzzi & Pasternak, 2002). The National Health & Nutrition Examination Survey (NHANES III Study) was conducted utilizing a representative sample of the population from 1988 to 1994 with a total of 6,149 participants ranging in age from 17-39 years. These participants were initially free of any cardiovascular diseases or chronic inflammatory conditions. The main predictor variable of interest was lifetime history of a major depressive episode assessed by the Diagnostic Interview Schedule. The main outcome variable was the presence or absence of an elevated CRP level ( $\geq 22$  mg/dl). The results showed that men with a history of depression were 2.77 times more likely to have elevated CRP levels than were those with no history of depression (95% confidence interval, 1.43-5.26). Furthermore, a history of major depressive disorder (MDD) was associated with elevated CRP, particularly for recent episodes, up to 6 months prior to the assessment. In women, a comparable association was weak and not significant (Danner, Kasi, Abramson, & Vaccarino, 2003).

Suarez (2004) examined the relationship between anger, hostility, and severity of

depressive symptoms, alone and in combination, and CRP levels in healthy men and women. The sample included 127 multiethnic, healthy, nonsmoking men and women. Fasting blood samples were collected the same day that assessments of anger and hostility (Buss-Perry Aggression Inventory-BPAQ) and depression (Beck Depression Inventory- BDI) were performed. A Psychological Risk Factor (PRF) score was generated as a composite summary indicator of BPAQ and BDI. This study confirmed associations in multivariate analyses that were statistically adjusted for various factors known to increase CRP such as gender, age, body mass index (BMI), alcohol use, exercise, and lipids. In analysis adjusting for these confounders, CRP was positively and significantly associated with a composite summary indicator of anger, hostility, and severity of depressive symptoms.

Ladwig, Marten-Mittag, Lowel, Doring, and Koenig (2005) assessed the possible combined effects of depressive mood (DM) and CRP as it may predict a future fatal or nonfatal coronary event. CRP and DM were analyzed in 3,021 apparently healthy male subjects aged 45-74. During a mean follow-up period extending 7.7 years, 165 CHD events occurred. CHD risks were estimated from Cox proportional hazard models and adjusted for age and survey and multiple risk factors. The age and survey adjusted interaction term of continuous CRP by DM showed a significant effect (HR 1.55; 95% CI 1.00-1.06;  $p = .037$ ). A stratified analysis of subpopulations with ( $n = 986$ ) and without ( $n = 2,035$ ) DM showed that high CRP ( $> 3\text{mg/L}$ ) was predictive in the group with DM (HR 2.69; 95% CI 1.32-5.47) but was not significant in the low-level depression group (HR 1.55; 95% CI 0.89-2.69). Interestingly, the low CRP/no depression subgroup ( $n = 565$ ) did not significantly predict a future CHD event. However, combined CRP and DM ( $n = 282$ ) significantly predicted future CHD events (HR 2.91; 95% CI 1.25-2.18;  $p = > .0001$ ). These results indicate that in apparently healthy men, a DM substantially increases the power of elevated CRP to predict subsequent cardiac event.

Dressler, Balieiro, Ribiero, and Dos Santos (2006) conducted a study on a Brazilian

population where there was a wide range of variation in both background characteristics (such as SES) and coronary artery disease risk factors. Fasting blood samples were obtained and evaluated for CRP in a sub sample ( $n = 258$ ) of individuals. A sample of 271 individuals was also interviewed using the Center for Epidemiological Studies Depression scale. The mean  $\pm$  SD CRP for the entire sample was  $0.43 \pm 0.44$  mg/L, with  $0.42 \pm 0.48$  mg/L for men and  $0.43 \pm 0.42$  mg/L for women. Data were analyzed using multiple regression analysis, controlling for age, sex, BMI, SES, tobacco use, and both total and LDL cholesterol. Higher reported depressive symptoms were correlated with higher CRP for men (partial  $r = -.298$ ,  $p = .004$ ) and with lower CRP for women (partial  $r = -.154$ ,  $p = .059$ ). According to the authors, this difference in men and women could be the result of differential effects of sex hormones on stress reactivity and immune response. Conversely, this difference in the associations could be related to gender differences in the disclosure of emotion and the effects that self-disclosure might have on physical health and immune response.

Ford and Erlinger (2004) estimated the odds ratio (OR) of elevated CRP level ( $> 0.21$  mg/ml) associated with depression in 6,914 non-institutionalized men and women ages 18-39 from the Third National Health and Nutrition Examination Survey (NHANES III). They found that the prevalence of lifetime major depression was 5.7% for men and 11.7% for women. A history of major depression was associated with elevated CRP level (OR, 1.64; 95% CI, 1.20 - 2.24). The association between depression and CRP was stronger among men than among women. The results were adjusted for age, African American race, BMI, log triglycerides, diabetes, systolic blood pressure, smoking status, alcohol use, estrogen use in women, aspirin use, ibuprofen use, and self-reported health status. Compared with men that did not have a history of depression, CRP levels were higher among men who had a more recent (within one year) episode of depression (adjusted OR, 3.00; 95% CI, 1.39 - 6.48) and who had recurrent ( $\geq 2$  episodes) depression (adjusted OR, 3.55; 95% CI, 1.55 - 8.14).

Douglas, Taylor, and O'Malley (2004) found conflicting results in a cross-sectional study of a cohort of 696, consenting, active duty US Army personnel undergoing a yearly physical. Depression was measured using the Patient Health Questionnaire (PHQ-9) and the Primary Care Evaluation of Mental Disorders (Prime-MD). A highly sensitive assay was used to measure CRP. The mean age of the group was 44 years ( $SD \pm 3$ ; 82% male). The mean CRP level was 1.7 mg/L (range, 0.3 - 9.9;  $SD \pm 1.6$  mg/L). Depression scores ranged from 0-26 with a mean of 2 ( $SD \pm 3$ ). Depression scores correlated positively with CRP levels ( $r = .0085$ ;  $p = .028$ ), as did other variables known to be associated with CRP like BMI ( $r = .36$ ), insulin levels ( $r = .22$ ), mean arterial pressure ( $r = .21$ ), triglycerides ( $r = .18$ ), exercise ( $r = -.12$ ), female sex ( $r = .097$ ), current smoking status ( $r = .08$ ), and high-density lipoprotein ( $r = -.09$ ). Interestingly, after controlling only for BMI, the relationship between depression and CRP lost statistical significance among women (adjusted  $r = .08$ ;  $p = .37$ ), among men (adjusted  $r = -.11$ ;  $p = .8$ ), and overall (adjusted  $r = .047$ ;  $p = .219$ ). Depressive symptoms seemed to only weakly correlate with CRP. The relationship between depression and coronary artery disease is unlikely to be explained through direct effects on CRP levels, but may be mediated by BMI. According to the authors, the results could possibly be explained due to the fact that the study was performed on a relatively young, physically fit group that may not be representative of the general population.

The research seems to indicate that depression has an exponential affect on CRP levels and particularly when the depressive episodes have occurred within a year prior to CRP levels being drawn (Douglas et al., 2004; Dressler et al., 2006; Ford & Erlinger, 2004; Ladwig et al., 2005; Suarez, 2004). Interestingly, in many studies, depression and elevated CRP were only associated with men and not women. Although there was an increase in CRP levels in women with depression, the difference was unable to achieve statistical significance (Douglas et al., 2004; Dressler et al., 2006; Ford & Erlinger, 2004; Saurez, 2004).

## Cortisol Levels

Research shows that depression can raise cortisol levels in the blood. High cortisol levels will increase endothelial inflammation by excess clotting. This can be associated with hypertension, increased cholesterol, and glucose dysregulation (Januzzi & Pasternak, 2002; Kemp et al., 2003).

Otte et al. (2004) tested cortisol levels in depressed CHD patients. Of 693 participants, 138 (20%) had depression at the time of the study ( $42 \pm 25$  vs.  $36 \pm 20$   $\mu\text{g/day}$ ,  $p < .01$ ). The odds of having depression increased twofold for participants with the highest levels of cortisol compared to those with the lowest levels of cortisol. This association remained strong even after adjusting for certain confounding variables (OR 2.4, 95% CI 1.3 - 4.4,  $p = < .01$ ).

Asnis et al. (1981) conducted a study on 25 unmediated hospitalized patients aged 26-64. These patients have severe MDDs and were evaluated clinically and endocrinologically by sampling plasma cortisol every 30 minutes for 24 hours. Measures of cortisol secretion did not correlate with symptom severity, but a relationship appeared between cortisol secretion and age during endogenous depressive illness. Following clinical recovery, as plasma cortisol levels moved back toward normal levels, there was no significant relationship between cortisol secretion and age. During depressive illness then, advanced age appeared to be an important factor as far as increasing cortisol levels but was of little significance as clinical recovery was achieved.

Preussner, Hellhammer, Preussner, and Lupien (2003), discovered that higher levels of depressive symptomatology were associated with greater cortisol levels after awakening. Forty healthy young men were assessed for severity of depressive symptoms using the Hamilton Depression Inventory and chronic and acute stress perception. The subjects were tested once a week, for 4 consecutive weeks, and provided saliva samples at 0, 30, and 60 minutes after awakening. The positive relationship between higher levels of depressive symptomatology and greater levels of cortisol seemed to be stronger in patients that were in the non-clinical range of

depression. Furthermore, depressive symptomatology and cortisol levels were significantly positively correlated with measures of chronic and acute stress perception.

Interestingly, Bhagwagar, Hafizi, and Cohen (2003) measured whether cortisol levels were elevated in fully recovered depressed patients. Thirty-one medication-free, recovered depressed patients and 31 matched healthy comparison subjects had salivary cortisol measured upon waking and at 15-minute intervals for the next hour. Their findings confirmed that greater secretion of cortisol may be present in depressed subjects even after clinical recovery and discontinuation of medication. This puts these patients at greater risk for recurrent episodes of depression as well as comorbid medical conditions such as coronary artery disease.

Although much research supports the idea that depressive symptomatology increases the level of cortisol in the bloodstream, there are some interesting differences discovered by various authors. Asnis et al. (1981) found a difference in raised cortisol levels based on advancing age, even though as the depression subsided, age was no longer a factor. Pruessner et al. (2003) found a positive relationship between depressive symptoms and increased cortisol levels, but the highest levels of cortisol were discovered in patients that were in the non-clinical range of depression. Bhagwagar et al. (2003) found that even after patients had clinically recovered from depression, their cortisol levels continued to be increased. It would appear that depression definitely raises cortisol levels and that increased cortisol levels cause inflammation in artery walls adding to an increased risk for coronary artery disease.

### **Risk Factors for Depression**

Research shows that there may be certain factors that influence the development of depression for the cardiac patient (Kemp et al., 2003; Lehto et al., 2000; Lesperance & Frasere-Smith, 2000; Thornton, 2001). A positive family history of depression is the most prevalent risk factor found in the literature (Lesperance & Frasere-Smith, 2000). Although psychological

stressors do not always indicate risk factors for depression, they can be a potential cause (Thornton, 2001). There are certain clusters of risk factors for depression that are also well-known risk factors for CHD and include: smoking, disability or poor (NYHA) class, stress and high workload, and history of CHD (Lehto et al., 2000). Social isolation and poor social support have been shown to increase an individual's risk for developing depression (Kemp et al., 2003; Lesperance & Frasere-Smith, 2000; Thornton, 2001). A newly emerging idea that is receiving a great deal of attention is called the type "D" personality. This personality type is considered a high-risk personality and is characterized by negative affectivity and social inhibition combined (Thornton, 2001). This personality type is rapidly replacing the highly regarded notion of the type "A" personality in regards to CHD. Each of these risk factors for depression will be discussed below.

### **Positive Family History of Depression**

Research has suggested that a positive family history of depression may have some impact on whether an individual will develop depression in his/her lifetime. In addition, family history of depression may give some important information about age of onset of depression. Due to its importance, a question regarding family history of depression is routinely asked by clinicians in a psychological exam (Nierenberg et al., 2007). This section will review the research conducted regarding the relationship between a positive family history of depression and its effects upon the development of depression.

Williamson, Birmaher, Axelson, Ryan, and Dahl (2004) conducted a study to examine the development of first-onset of MDD in children either coming from high familial risk of depression or low familial risk of depression. Those children that were free of any psychiatric disorder, that had at least one first degree and one second degree relative with a lifetime history of childhood-onset, bipolar, recurrent, or psychotic depression were included in the high risk group



( $n = 76$ ). Those children that were free of any lifetime psychiatric disorders and had no first degree and less than 20% of second degree relatives with a lifetime affective disorder were included in the low risk group ( $n = 63$ ). Using the Schedule for Affective Disorders and Schizophrenia for School Age Children-Epidemiologic version (K-SADS-E), children and their parents were assessed. Follow-up interviews were conducted, on average, 18 months apart and extended for 6 years. The results showed that high-risk children had an approximate threefold increased risk of developing a first onset MDD compared with their low-risk counterparts (OR = 3.21). The average age of new-onset MDD was 14.0  $\pm$  2.9 years with a range of 9.5 – 19.5 years. High familial loading for affective disorders, a mother with depression or anxiety disorder, and a behavioral disorder in the child significantly contributed to the risk for developing depression in the future.

Furthermore, Tozzi et al. (2008) conducted a study to determine if the presence of family history of depression or anxiety, in a first-degree relative, caused a younger age of onset for depression. A sample of 1,022 cases with recurrent MDD was recruited from the Max Planck Institute and two affiliated hospitals. The Schedules for Clinical Assessment in Neuropsychiatry and questionnaires included demographic information, medical history, alcohol and tobacco use, personality traits, and life events. Survival analysis and the Cox proportional hazard model were used to determine if a family history of depression could indicate an earlier age of onset of depression. The results showed that of those patients that reported a positive family history of depression, there was a significantly earlier age of onset of depression than in patients that did not report a family history of depression (log-rank = 48,  $df = 1$ ,  $p < .0001$ ). The magnitude of the association between family history and depression varies according to age of onset, with the largest estimate for MDD onset prior to the age of 20 years (hazard ratio = 2.2,  $p = .0009$ ), whereas family history is not associated with MDD when the onset occurs after the age of 50 years (hazard ratio = .89,  $p = .5$ ). Interestingly, feelings of guilt and anxiety, as well as functional

impairment due to depression, appear to characterize those individuals with a positive family history of depression. Therefore, a family history of depression appears to contribute to an earlier age of onset of depression, and may also affect the clinical features of the disorder.

Nierenberg et al. (2007) set out to discover if patients who have MDD and a positive family history are different from those patients that have MDD but no family history of depression. Demographic and clinical features were compared from a large cohort of patients seeking outpatient treatment with non-psychotic MDD who had reported that they had or did not have at least one first-degree relative who had MDD or bipolar disorder. All subjects ( $n = 4,005$ ) were recruited from the STAR (\*) D multi-center trial. The differences in demographics and clinical features for the patients with and without a family history of mood disorders were assessed after correcting for sex, age, race, and ethnicity. Of those patients that had a positive family history of mood disorder ( $n = 2,265$ ; 56.5%), they were more frequently women and typically had an earlier age of onset of depression as opposed to those patients that had no family history of mood disorder ( $n = 1,740$ , 43.5%). No meaningful differences were found in depressive symptoms, recurrence, severity, daily function, and depressive subtype.

Mondimore et al. (2006) conducted a study to determine if a chronic course of depression could define a familial clinical subtype in MDD. A measure of lifetime chronicity of depressive symptoms such as recurrent mood symptoms most or all of the time, was tested in 638 pedigrees for familial aggregation, using the Genetics of Recurrent Early-Onset Depression (GenRED) project. The findings showed that in subjects with chronic depression, the mean age at depressive illness was lower, and that the rates of attempted suicide, panic disorder, and substance abuse were higher in those subjects with non-chronic depression. Interestingly, chronicity was assessed in 37.8% of affected first-degree relatives of subjects with chronic depression, and in 20.2% of relatives of subjects that had non-chronic depression. Analysis that used the generalized estimating equation model showed ( $OR = 2.52$ ;  $SE = .39$ ,  $z = 6.02$ ,  $p < .0001$ ) for the likelihood

of chronicity in a subject predicting chronicity in an affected relative. The findings suggest that the chronicity of depressive symptoms is familial, especially in preadolescent onset illness.

Puig-Antich et al. (1989) assessed first-degree ( $n = 195$ ) and second-degree ( $n = 785$ ) adult relatives of pre-pubertal children with major depression ( $n = 48$ ). Participants were assessed by using the Family History-Research Diagnostic Criteria method (FH-RDC). Compared to normal control children, pre-pubertal children with MDD had significantly higher familial rates of psychiatric disorders in both first and second-degree relatives, especially MDD, alcoholism, and “other” diagnoses (mostly anxiety). Likewise, relatives of children in the non-affective psychiatric control (PC) group had lower rates of alcoholism, but higher rates of other (anxiety) disorder diagnoses, and intermediate rates of MDD. The research suggests that pre-pubertal onset of MDD may be especially likely in families with a high incidence of affective disorders when these particular families also have a high rate of alcoholism, and that a proportion of children without affective disorder but with separation anxiety were at high risk for the development of affective illness in their later years of life.

Klein, Lewinsohn, Seeley, and Rohde (2001) felt that family studies were a useful way to explore continuities and discontinuities between MDD amongst children and adolescents and MDD in adults. They conducted a family study of MDD in a large community sample of adolescents. The study included 268 adolescents with a history of MDD, 110 adolescents with a history of non-mood disorders, but no history of MDD through the age of 18 years, and 291 adolescents with no history of psychopathology through 18 years. Psychopathology was then assessed in 2,202 first-degree relatives of all the subjects. This was assessed through semi-structured direct and indirect family history interviews. The best estimate diagnoses were calculated by using all available data. The findings showed that the relatives of adolescents with MDD showed significantly elevated rates of MDD (hazard ratio [HR], 1.77; 95% CI, 1.46-2.31), dysthymia (HR, 1.79; 95% CI, 1.11-2.87), and alcohol abuse or dependence (HR, 1.29; 95% CI,

1.05-1.53). The results provide support that familial aggregation of depressive symptoms increases adolescent MDD.

A familial history of depression appears to be a strong predictor of depressive symptoms, especially when the history appears in first and second-degree relatives. It is very difficult to find any research to dispute the link between familial history and depression to some degree. Even more evident is the fact that a family history of depression affects the age at onset. The greater the family history, the earlier the onset of symptoms seems to appear (Klein et al., 2001; Mondimore et al., 2006; Puig-Antich et al., 1989; Tozzi et al., 2008; Williamson et al., 2004).

### **Psychological/Life Stressors**

There appears to be a causal relationship between stressful life events and major depression. This relationship may also be influenced by genetic factors that predispose to specific reactions to psychological and life stresses (Kendler, Karkowski, & Prescott, 1999).

Kendler et al. (1999) assessed the occurrence of 15 classes of stressful life events and the onset of DSM-III-R major depression over a one-year period in female twins extrapolated from a population-based registry. The sample included 24,648 person-months and 316 onsets of major depression. The stressful life events were rated individually on contextual threat and dependence (the degree to which the stressful life event could have resulted from the respondents behavior). The nature of the relationship between major depression and stressful life events was tested by discrete-time survival analysis that examined the relationship between dependence and the depressive effect of stressful life events and a co-twin control analysis. The results showed that while independent stressful life events were significantly associated with individual onsets of depression, if the level of threat was controlled, the association was significantly stronger for dependent events. The odds ratio (OR) for the onset of major depression during a month of a stressful event was 5.64 in all subjects, 4.52 within dizygotic pairs, and 3.58 within monozygotic

pairs. Stressful life events appear to have a substantial causal relationship with the onset of episodes of depression. Interestingly, about one-third of the association between stressful events and onsets of depression is non-causal, since individuals predisposed to major depression often select themselves into high-risk environments.

Marinic, Ozvacic, Katic, Polasek, and Lazic (2007) conducted a study on post-Croatia War (1991-1995) survivors in a town called Otocac, as this town was deeply affected by the effects of the war. The study was conducted ten years after the war. The goal of the study was to evaluate the prevalence of depression in general practice patients ten years following a major life-altering stressful event, such as war. In 2005, 300 patients were picked randomly and systematically selected from a patient listing of a general practitioner in Otocac, Croatia. All subjects were 18 years or older. Using the ICD-10 classification of Mental and Behavioral Disorders, the patients were classified as having mild, moderate, or severe depressive episode with psychotic elements. The differences in the characteristics of depressed and non-depressed patients were tested using the chi2-test and Fisher's test. The results showed that a total of 147 (49%) of patients (65 male and 82 female) met the ICD-10 criteria for depressive episode. The distribution of severity of depression showed that the majority of depressed patients met the criteria for moderate (38.8%) and severe (39.5%) depression.

Chen et al. (2007) conducted a study to examine quality of life (QOL), prevalence of posttraumatic stress disorder (PTSD), and depression in firefighters in Kaohsiung, Taiwan. These firefighters experience an enormous amount of emotional shock from disasters, which could result in psychiatric impairments. This study involved a two-stage survey. In the first stage, 410 firefighters were given the 36-item Short-Form Health Survey (SF-36) and the Disaster-Related Psychological Screening Test (DRPST) to assess QOL, possible PTSD, possible major depression, and related risk factors. In the second phase, psychiatrists categorized the cases based on the self-report questionnaires that were completed and separated, according to DSM-IV into

PTSD or major depression group, subclinical group, and health group. The results showed that prevalence rates for major depression and PTSD were 5.4% (22/410) and 10.5% (43/410), respectively.

Coruuble (2006) conducted a study to examine the relationship between the number of prior episodes of depression and the stressful life event exposure that triggered the current depressive episode. There were interesting relationships that were discovered such as: the linear increase of the number of depressive episodes with increasing age, severity of depression, female gender, and family history of depression. The most important relationship discovered, however, was that as the number of previous depressive episodes increased, the number of stressful life event exposures decreased, triggering a depressive episode. As the number of previous depressive episodes increased, it was discovered that even minor stressful life events could trigger a new depressive episode.

Amital (2008) further hypothesized that of those patients that suffer from treatment resistant depression (TRD), negative/stressful life events could be an independent risk factor for TRD. Subjects consisted of 107 unipolar MDD patients. These patients had all been treated for at least 4 weeks. The patients were assessed on their psychiatric and medical history and a total of seven categories of stressful life events. The results showed that 39.3% of the subjects were defined as TRD patients and 60.7% as non-TRD patients. TRD patients had more severe depression, greater suicidal attempts, longer episodes of depression, more hospitalizations, more benzodiazepine usage, and more antipsychotic usage. Job stress and financial stress were more prevalent in the TRD group.

The research seems to show that stressful events play a role in the precipitation of depression. It is also possible that depression might evolve as a process of several neuronal changes that occur in several brain regions. It is likely that these stressors could elicit neurochemical changes, possibly due to genetic determinants, and a wide array of environmental

and experiential factors (Anisman, Merali, & Stead, 2008).

Elovaino et al. (2007) studied the role of DRD2 polymorphism (rs1800497) in the association between depressive symptoms and stressful life events. Stressful life events are often associated with negative health outcomes, but it is apparent that there are large individual differences when it comes to coping with these events. Variants in dopamine receptor genes, such as DRD2, are associated with depression, but it is not clear if these variants can also modify the association between life events and depression. Prospective data were analyzed on life events and depressive symptoms in 1992 and 2001 that related to 1,611 young adults (672 men and 939 women, aged 15-30). These subjects participated in the ongoing population-based cardiovascular risk in young Finns study. The results showed that depressive symptoms did increase as the number of stressful life events increased. However, the association was seen only in those subjects that carried A2/A2 ( $n = 872$ ) genotype. The association was not detected in subjects that carried the A1/A1 or A1/A2 ( $n = 486$ ) genotype. It seems that DRD2 polymorphism can moderate the effect of stressful life events on depressive symptoms. Furthermore, those individuals that carry the A2/A2 DRD2 genotypes may be more vulnerable to depression than others may.

Cervilla et al. (2007) conducted a study to test the potential gene-by-environment interaction between 5HTTLPR genotype at the serotonin transporter gene along with previous exposure to threatening life events (TLE) in regards to depression. There were a total of 737 participants that were genotyped. Information was gathered regarding exposure to TLEs over the last six months, sociodemographic information, and family history of psychological problems among first-degree relatives. Trained interviewers used The Composite International Diagnostic Interview (ICD) to diagnose depression. Two outcomes were used: the ICD-10 depressive episode, and ICD-10 severe depressive episode. Both the s/s genotype and the exposure to increased numbers of TLE's were significantly associated with depression. Furthermore, the

5HTTLPR s/s genotype significantly modified the risk associated with TLE's for both depressive outcomes. The s/s homozygous participants only needed minimal exposure to TLE (1) to increase their risk for depression. The same risk was only found in l/s or l/l individuals after a higher exposure to TLEs (2 or more).

Diener, Kuehner, Brusniak, Struve, and Flor (2008) studied the effects of uncontrollability and helplessness in the face of stressful life events as an important determining factor in regards to developing and maintaining depression. It has been suggested that the inability to deal with stressful life events could be related to dysfunctional prefrontal lobe functioning in the brain. Cognitive, behavioral, and physiological effects of stressor uncontrollability in depressed and healthy individuals were assessed. Relationships between altered cortical processing and cognitive vulnerability were also assessed. Twenty six unmedicated depressed patients and 24 matched healthy controls were tested in an expanded forewarned reaction (S1-S2) paradigm. Using a factorial design, stressor controllability varied across three conditions: (a) control, (b) loss of control, (c) restitution of control. Error rates, ratings of controllability, arousal, emotional valence, and helplessness were assessed throughout the experiment with the post-imperative negative variation (PINV) of the electroencephalogram. The results showed that depressed patients showed an enhanced frontal PINV during both loss of control and restitution of control. These patients also felt more helpless than the controls. The findings seem to indicate that depressed patients are more susceptible to stressor uncontrollability than their healthy control counterparts. The alterations in prefrontal functioning appear to add to this vulnerability and, therefore, can be linked to trait markers for depression.

The research found a wide variety of studies that seemed to find many explanations to the relationship between psychological/life stressors and depression. Kendler et al. (1999) found that independent stressful life events were significantly associated with individual onsets of depression, but that if the level of threat could be controlled, the association was significantly



stronger for dependent events. Corruble (2006) discovered that as the number of previous depressive episodes increased for an individual, the number of stressful events needed to trigger a depressive episode decreased. For those with TRD, negative/stressful life events could even be considered an independent risk factor for TRD (Amital et al., 2008). Furthermore, research has delved into the concept that life stressors could elicit neuro-chemical changes, possibly due to genetic determinants such as DRD2 polymorphism (Anisman et al., 2008; Elovaino et al., 2007). Cervilla et al. (2007) studied the gene-by-environment interaction as well. Uncontrollability and helplessness in the face of stressful events seems to make individuals more susceptible to depression (Diener et al., 2008). Although these ideas seem to be diverse, the general consensus seems to be pointing in the direction that stressful life events do increase an individual's risk for developing depression.

### **Smoking**

There are several hypotheses associating smoking status as a risk factor for depression. First, according to the self-medication hypothesis, those who suffer from depressive symptoms use smoking to alleviate depressive symptoms. The second assumption asserts that chronic smoking may play a role in the etiology of depression. The third assumption suggests that there is a reciprocal mechanism between smoking and depression. The last assumption asserts that there are shared underlying genetic factors explaining this comorbidity (Lyons et al., 2008)

Almeida and Pfaff (2005) investigated the association between smoking and depression in people aged 60 years and older. A cross-sectional survey was administered to a sample of general practitioners patients in Western Australia. One thousand and thirty patients completed the survey and were divided into four groups according to smoking status: never smoked, ex-light smoker, ex-heavy smoker (> 20 cigarettes/day), and current smoker. Depressive symptoms were then assessed with the Center for Epidemiologic Studies-Depression Scale (CES-D). A CES-D

score of 22 or more was used to define the presence of clinically significant depression. The participants ranged in age from 60-101 years and 57.2% of participants were female. The mean CES-D score for never smokers was  $10.5 \pm 8.3$ , for ex-heavy smokers it was  $12.5 \pm 10.2$ , and for current smokers it was  $13.1 \pm 11.0$ , ( $p = .037$ ). Current or past heavy smoking was associated with increased risk of clinically significant depression when compared to never or past light smoking (OR = 1.58, 95% CI, 1.01-2.48) after adjusting for age, gender, place of birth, social isolation, self-perceived health, and harmful or hazardous drinking. Past heavy smoking and current smoking were associated with increased frequency and severity of depression.

Lyons et al. (2008) conducted a study to examine the relationship between lifetime major depression, smoking, and nicotine dependence. There were 8,169 male twin subjects from the Vietnam War era. Twin registry biometrical modeling demonstrated a genetic influence on nicotine dependence, daily smoking, and major depression, and a family environmental influence on daily smoking. Genetic factors that influenced nicotine dependence also influenced major depression. Major depression was associated with current daily smoking as well as with certain nicotine withdrawal symptoms. Subjects that had a familial vulnerability towards major depression, even without a personal history of major depression, were more likely to smoke despite a serious illness and were more prone to report restlessness, nervousness, difficulty concentrating, and depressed mood during past attempts to quit smoking.

Munafo, Hitsman, Rende, Metcalfe, and Niaura (2008) aimed to investigate the relationship between smoking status and continuous depressed mood within a cohort of adolescents. A subset of adolescents was chosen that reported never having smoked a cigarette at baseline, some of whom progressed to smoking at a follow up one year later. Data were drawn from the National Longitudinal Study of Adolescent Health, an ongoing study to assess the health status of adolescents, to explore the causes of adolescent health-related behavior ( $n = 12,149$ ). The subsample that reported never having smoked a cigarette at baseline ( $n = 5,475$ ) was, on

average, 15 years old. The CES-D was used to measure depressed mood. Various findings showed relationships between smoking status and depressed mood, with a general trend for these effects to be greater in females. Among “never smokers” at baseline, level of depressed mood at baseline predicted subsequent progression to smoking initiation ( $p = .022$ ), but not progression to regular smoking ( $p = .229$ ). Interestingly, among “never smokers” at baseline, progression to smoking initiation during the follow-up period was associated with higher CES-D scores at follow up, even after adjusting for baseline depressed mood ( $p < .001$ ), with this effect being greater for females than males. Among subjects who initiated smoking, progression to regular smoking was associated with higher CES-D scores for follow up among females ( $p = .966$ ). The relationship between cigarette smoking and depression may be a factor in the development of subsequent dependence.

Husky, Mazure, Paliwal, and McKee (2008) tried to determine if smoking behavior was associated with current or lifetime major depression and whether the association was greater in women. Data were extrapolated from the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC: wave 1, from 2001-2002,  $n = 42,565$ ). Logistic regression was used to examine the relationship between smoking status (daily, occasional, prior) and Diagnostic and Statistical Manual IV (DSM-IV) major depression (current and lifetime) by gender in terms of odd ratios. The results showed that current (daily, occasional) and prior smoking significantly increased the odds of having current or prior major depression. Women with prior smoking history were at a significantly higher risk of current or past depression than were men (OR: 1.53 vs. 1.36; 1.72 vs. 1.36), as was true for current occasional (OR: 1.92 vs. 1.39; 1.90 vs. 1.30) and daily smoking (OR: 2.52 vs. 1.95; 1.84 vs. 1.48). It appears that the association between smoking and current or past depression is not necessarily limited to smoking that meets the criteria for nicotine dependence, and is more potent in women.

The literature seems to suggest that individuals that currently smoke or have a past

history of smoking are at greater risk for depression than their nonsmoking counterparts (Almeida & Pfaff, 2005; Lyons et al., 2008). Furthermore, the risk for depressive symptoms seems to be even greater in women than it is in men, whether they are current smokers or past heavy smokers (Husky et al., 2007; Munafo et al., 2008).

### **History of CHD/MI**

Following a cardiac event such as an MI, depression can become very common and can develop anywhere from 3, to 12, to even 18 months after the event. Depression after an MI can have influence on QOL, medical compliance, and cardiological prognosis (Krzyzkowiak, 2007).

Lehto et al. (2000) studied the prevalence of depression at least six months after various CHD events and the association between depression and certain clinical variables. In their study, 414 patients (284 males, 130 females) younger than 71 years (mean age for men 60.9 years, and for women 63.6 years) were interviewed and examined for smoking habits, BMI, lipid levels, and diabetic status. The Depression Scale (DEPS) was used to screen patients for depression. The NYHA Class was also assessed. Among these groups, one-sixth, or 14-19% of the patients, suffered from depression.

Romanelli, Fauerbach, Bush, and Ziegelstein (2002) conducted a study four months after a patient was discharged from the hospital with certain cardiac diagnoses. Of the participants, ( $n = 284$ ) who had an acute MI, 153 (53.9%) of these were aged 65 years and older. One hundred and one of these (66.0%) actually completed the four-month follow-up interview. One of the findings was that older patients with depression were more likely to have had a prior MI than older patients without depression (54.3% vs. 31.0%,  $p = .012$ ).

Dickens et al. (2004) conducted a study to consider whether the causes of depression occurring before and after MI were similar to those of depression in the general population. Consecutive patients admitted to the hospital following a first MI were interviewed with the

Schedule for Clinical Assessment in Neuropsychiatry in order to detect psychiatric disorders and the Life Events and Difficulties Schedule to assess recent stress. Patients also completed the Hospital Anxiety and Depression Scale (HADS) at entry to the study and then one year later and the risk factors associated with a high score at both times were assessed. Of 314 patients (88% of those eligible) that were recruited, 199 (63%) were male and 63 (20%) had depressive disorders. Logistic regression identified the following as independently associated with depressive disorder that had been present for at least one month prior to the MI: younger age, past psychiatric history, female gender, social isolation, and lack of a close confidant regarding marked non-health difficulties. Upon follow up, 269/298 (90%) had responded; of 189 participants that were not depressed at the first assessment, 39 (21%) became depressed at the one-year follow up. Logistic regression identified frequent angina as the only significant predictor of a raised HADS score at 12 months. The risk factors that precede an MI may not be the same as those that come after an MI, but a link definitely exists connecting the development of depression following an MI.

Krzyzkowiak (2007) conducted a study to assess depressive symptom intensity after an MI, amongst other variables. The Mini International Neuropsychiatric Interview, BDI, Social Readjustment Rating Scale, and Recent Life Changes Scale were used to assess depression in 102 patients following MI. Of these patients, 10.8% were diagnosed with clinical (major) depression. However, depressive symptoms were present in 40%.

Kaptein et al. (2006) conducted a study to identify the presence of depressive symptoms in patients after an MI. Data were retrieved from the Depression after Myocardial Infarction (DepreMI) study, a naturalistic follow-up study of patients that were admitted to four hospitals in the Netherlands for MI ( $n = 475$ ). The BDI was administered during hospitalization, and then at 3, 6, and 12 months post-MI. The prevalence of significant depressive symptoms ranged from 22.7% to 25.5% throughout the post-MI year. Five distinct courses were identified: no depressive symptoms (56.4%), mild depressive symptoms (25.7%), moderate and increasing depressive

symptoms (9.3%), significant but decreasing symptoms (4.6%), and significant and increasing depressive symptoms (4.0%).

Parakh, Thombs, Fauerbach, Bush, and Ziegelstein (2008) conducted a study to determine if depression during hospitalization for MI was associated with mortality and if this risk persisted over a longer period of time. Major depression and dysthymia were assessed using the structured interview for Diagnostic and Statistical Manual of Mental Disorders, Revised, Third Edition, and depressive symptoms, using the BDI. The subjects included 284 patients that were hospitalized for MI. Any type of depression (major, dysthymia, and/or BDI score of  $\geq 10$ ) was discovered in 76 (26.8%) patients.

Based on the literature, it appears as though depression is very common in patients that have experienced an MI. Throughout the studies, it seems as though the percentages of depressed patients is consistently above 20% (Dickens et al., 2004; Kaptein et al., 2006; Krzyzkowiak, 2007; Parakh et al., 2008). Even more importantly, the course of depression does not always diminish over time. In many cases the course of depression increases. In some cases those that were not depressed at the time of discharge from the hospital were found to be depressed at their one-year follow up (Dickens et al., 2004).

### **Social Isolation/Social Support**

Depression and social isolation/poor social support affect one in seven people over the age of 65. There is increasing evidence that these factors can adversely affect long-term health. Interventions that promote social contact, and encourage creativity are likely to promote better health and overall well-being (Januzzi & Pasternak, 2002).

Greaves (2006) conducted a study to evaluate a complex intervention for addressing social isolation in older people. Mentors delivered a series of individually tailored activities. These activities were tapered off over time. Two hundred and twenty-nine participants at the

Upstream Healthy Living Center were offered the Geriatric Depression Scale (GDS), SF12 Health Quality of Life, and Medical Outcomes Social Support Scale at baseline. These same assessments were offered at 6 and 12 months post intervention. Data were available for 172 (75%) participants at baseline, 72 (53% of those eligible) at 6 months and 51 (55%) at 12 months. Baseline scores indicated social isolation and high morbidity for physical and mental health. The intervention was successful in engaging this population (80% participated in some form of activity). At 6 months there were significant improvements in the SF12's mental component and the depression scores, but not in perceived physical health or social support. At 12 months, there were significant improvements in depression and social support but only a marginally significant improvement in SF12's physical component ( $p = .06$ ); however, the SF12 mental component was not maintained. Interestingly, the data showed a wide range of responses, both emotional and physical. The responses included increased mental alertness, social activity, self worth, optimism about life, and positive changes in health behavior.

Xie et al. (2005) studied the effects of peer isolation and social support availability on the relationship between BMI and depressive symptoms. Baseline data were gathered from 1,655 Chinese adolescents between 11 and 15 years of age. Information was retrieved from a longitudinal smoking cessation and health promotion program in Wuhan, China. Assessments of height and body weight, depressive symptoms, perceived peer isolation (PPI), and perceived availability of social support (PASS) were gathered. According to the International Obesity Task Force (IOTF) age and sex specific BMI cutoffs, 12.5% of the boys and 9.2% of the girls were overweight. Among the girls, high BMI was significantly related to higher self-reported depressive symptoms, and was primarily mediated by PPI. On the contrary, high BMI boys reported significantly lower levels of PPI, although high PPI level definitely increased depressive symptoms. In both boys and girls, the observed effect of PPI on the relationship between BMI and depressive symptoms was only sustained in low-PASS girls and boys. This study reveals that

there are differences in boys and girls relating to the effects of PPI on the association of BMI and depressive symptoms, which was buffered only by levels of PASS.

Vanderhorst and McLaren (2005) studied the human relatedness of variables such as social support resources, marital status, and sense of belonging as predictors of depression and suicidal ideation in older adults. A community sample of 110 older adults with a mean age of 76.67 years,  $SD = 8.11$ , was used. They each completed the Social Support Subscale of the Coping Resources Inventory, the Sense of Belonging Instrument, the Zung Depression Inventory, and the Suicide Subscale of the General Health Questionnaire. The results showed that fewer social support resources were associated with higher levels of depression and suicidal ideation. Interestingly, the researchers found that sense of belonging was not a good predictor of mental health.

Stephens, Owen, Kunz-Ebracht, and Brydon (2004) conducted a study to determine if loneliness, which is related to social isolation and perceived lack of companionship, is a health risk. The revised UCLA loneliness scale was completed by 240 working men and women aged 47-59 years. This scale was related to affective state and cardiovascular, neuro-endocrine, and inflammatory processes. The loneliness scores were not associated with age, gender, or socioeconomic status, but were lower in married participants than in divorced or single participants. These scores were positively related to social isolation, low emotional support, hopelessness, ratings of depression, low self esteem, and sleep problems. Lonely individuals displayed a significantly higher level of fibrinogen ( $p = .038$ ). The cortisol levels in lonely individuals were elevated within the first 30 minutes after awakening, even after adjusting for waking cortisol value, sex, socioeconomic status, smoking, time of waking, and body mass ( $p = .046$ ). The authors concluded that loneliness is a psychological experience that has potentially negative effects on our biological stress responses and could very well be detrimental to one's health.



Wang, Mittleman, Leineweber, and Orth-Gomer (2006) conducted a study to examine the joint effects of depressive symptoms and social isolation regarding the progression of atherosclerosis in women. There were 102 women enrolled in the Stockholm Female Coronary Angiography Study in Sweden between 1991 and 1994. Coronary atherosclerosis progression was evaluated by using a computer-assisted standardized assessment. This assessment looked at the mean luminal diameter change in the artery over 3 years in a series of 10 predefined coronary segments. Social isolation and depressive symptoms were assessed by using standard questionnaires. Multivariable controlled mixed model ANOVAs showed that women who were both depressed and socially isolated had the greatest amount of disease progression. Their absolute mean luminal diameter decreased by 0.18 mm (95% CI = .11-.24) and their percent narrowing was 5.5% (95% CI = 3.6-7.4). In women who had neither psychological factor, the mean luminal diameter decrease was 0.04 mm and their narrowing was 0.9%. It would appear that in women with coronary disease, depressive symptoms, and social isolation combined, the disease process was accelerated. This seems to suggest a direct psychological effect on the atherosclerotic process.

Choi and McDougall (2007) conducted a study to compare homebound older adults to their ambulatory peers who attended senior centers with respect to their depressive symptoms, depression risk and protective factors, and self-reported coping strategies. There were 81 low-income, homebound older adults, compared to 130 ambulatory older adults that attended a local senior center. Even when controlling for socio-demographics, health problems, and other life stressors, being homebound, as compared to attending a senior center, was significantly associated with higher depressive symptoms. Interestingly, however, when coping resources—social support and participation in frequent physical exercise—were added to the regression model, the homebound situation was no longer a significant factor.

Boden-Albala, Litwak, Elkind, Rundek, and Sacco (2005) conducted a study to assess the

relationship between social isolation and stroke outcomes in a multiethnic cohort. The authors prospectively followed 655- ischemic stroke cases for 5 years. The group consisted of 55% women, 17% white, 27% African American, 54% Hispanic, and a mean age of 69 +/- 12 years. These patients were enrolled in the Northern Manhattan Stroke Study. Baseline data were collected regarding social isolation. At their follow-up, the authors documented events as defined by the first occurrence of MI, stroke recurrence, or death. Cox hazard models were used to calculate the hazard ratio (HR, 95% CI) for pre stroke predictors for post stroke outcomes. The results showed that there were 265 first-outcome events. Using univariate analysis, coronary artery disease (OR 1.3, 1.0-1.7), age >70 years (OR 1.9, 1.5-2.5), atrial fibrillation (AF) (OR 1.8, 1.3-2.5), race-ethnicity (white vs. Hispanic) (OR 1.7, 1.1-2.9), physical inactivity (OR 1.3, 1.1-2.6), help at home (OR 1.8, 1.4-2.4), and social isolation (OR 1.4, 1.2-1.6) were associated with an increased risk of an outcome event. It appears that pre stroke social isolation is a predictor of outcome events post stroke.

Schiffer et al. (2005) aimed their study to discover if Type D personality was associated with an impaired health status and increased depressive symptoms in patients with CHF, independent of disease characteristics. Patients ( $n = 84$ , 63 men and 21 women), with systolic CHF completed four different questionnaires to assess Type D personality (14-item Type D Personality Scale [DS14]), health status (Minnesota Living with Heart Failure Questionnaire [MLWHFQ]), depressive symptoms (Center for Epidemiological Studies-Depressions [CES-D]), and mood status (Global Mood Scale [GMS]) upon visiting an outpatient heart failure clinic. Patient medical charts were also utilized to obtain certain other clinical variables. The results showed that Type D patients were more likely to experience impairment in health status ( $18/38 = 47\%$ ) as compared to non Type Ds ( $11/46 = 24\%$ ), ( $p = .027$ ). These same Type D patients also reported more depressive symptoms than the non-Type Ds ( $18/38 = 47\%$  vs.  $6/46 = 13\%$ ,  $p = .001$ ). Even when controlling for severity and etiology of CHF, age, and gender, Type D

continued to be a significant associate of impaired health status (OR 3.0, 95% CI, 1.12-7.78) and depressive symptoms (OR 6.3, 95% CI, 2.08-19.12).

Karlsson et al. (2007) further discovered that if Type D patients were given more expanded cardiac rehabilitation (stress management, increased physical training, staying at a “patient hotel” following discharge, and cooking lessons), the outcome was significant. The study randomized 224 cardiac patients either to an expanded cardiac rehabilitation program or to a traditional, routine cardiac rehabilitation program. These patients were followed up on within one year. At baseline, the patients that had a high Type D score, had a lower sense of coherence ( $p < .001$ ), a lower QOL ( $p < .001$ ), more depressive symptoms ( $p < .001$ ), and increased anxiety ( $p < .001$ ) as compared to those patients with a low Type D score. After follow up, the Type D patients that had been randomized to the expanded cardiac rehabilitation program showed significant decreases in their Type D scores ( $p < .01$ ), depression and anxiety ( $p < .05$ ), and an increment in QOL scores ( $p < .001$ ). It would appear that an expanded cardiac rehabilitation program could have a significant effect on patients that have a Type D personality and improve anxiety, depression, and QOL.

Given the great amount of research that has been conducted in regard to Type D personality and its association to impaired health status (particularly for further risk of CHD), it would appear that a great deal of value ought to be placed on including the assessment of personality factors when evaluating the cardiac patient (Schiffer et al., 2005).

### **Type D Personality**

Type D personality refers to the tendency to experience negative emotions and to inhibit the expression of these emotions in social interactions or relationships (Denollet, Pedersen, Vrints, & Conraads, 2006). The “D” stands for “distressed.” John Denollet, a Dutch professor of medical psychology, is the founder of this concept. Research has shown that CHD patients with a

Type D personality have a worse prognosis after an MI as compared to patients without the Type D personality. Type D personality is associated with four to eight times the increased risk of mortality, recurrent MI, or sudden death (Denollet et al., 2006). Type D personality has also been associated with increased risk of depression, exhaustion, and social alienation (Pedersen & Denollet, 2004).

Denollet et al. (1996) postulated that the Type D personality was an independent predictor of long-term mortality in CHD. To test this hypothesis, 268 men and 35 women with angiographically proven CHD, aged 31-79 years, who were taking part in an outpatient cardiac rehabilitation program, were studied. Each patient completed a personality questionnaire at entrance to the program. These same patients were contacted 6-10 years later to establish survival status. The main endpoint was death from all causes. The results showed that 38 patients had died, and of those, 24 were cardiac related deaths. The rate of death was statistically significantly higher for Type D patients than for non-Type D patients (33 [27%]/85 vs. 15 [7%]/218;  $p < .00001$ ). Evidence of the association between Type D personality and mortality was still strong more than 5 years after the coronary event in both men and women. In this study, mortality was associated with impaired left ventricular function, three-vessel disease, low exercise tolerance, and the lack of thrombolytic therapy after an MI. Even when controlling for these biomedical predictors, using the multiple logistic regression analysis, the impact of Type D personality remained significant (OR: 4.1, 95% CI, 1.9 -8.8,  $p = .0004$ ). Social alienation and depression were also related to mortality, but did not have the predictive power of the Type D personality.

Denollet et al. (2006) investigated the relative effect of stress and Type D personality relating to prognosis at 5-year follow ups. Three hundred and thirty seven patients with CHD who were participating in a cardiac rehabilitation program were studied. Patients filled out the General Health Questionnaire (to determine psychological stress) and the Type D Personality Scale. Patients were followed for 5 years. The end point consisted of major adverse cardiac events.

There were 46 major adverse cardiac events at follow up that included four deaths and eight MIs. Patients with Type D personality had an increased risk of death/MI (OR: 4.84, 95% CI, 1.42-16.52,  $p = .01$ ) compared to non-Type D personality patients, independent of disease severity. Stress ( $p = .011$ ) and Type D ( $p = .001$ ) were related to increase the risk of developing a major adverse cardiac event even after adjusting for gender, age, and biomedical risk factors. Multivariate analysis showed left ventricular ejection fraction (EF)  $\leq 40\%$ , no treatment with CABG, and Type D personality as independent predictors of major adverse cardiac events (OR: 2.90, 95% CI, 1.4-5.92,  $p = .003$ ), whereas psychological stress was only marginally significant (OR: 2.01, 95% CI, 0.99-4.11,  $p = .054$ ). Type D personality may well be added to the risk stratification of CHD patients.

Denollet, Vaes, and Brutsaert (2000) hypothesized that chronic emotional stress creates an increased risk for negative outcomes regardless of appropriate treatment. This prospective study examined the five-year prognosis of 319 CHD patients. Assessments for depression/anxiety and Type D personality were administered at baseline. The main end points included cardiac death or nonfatal MI and impaired QOL. Twenty-two cardiac events (nonfatal) occurred. These events were related to left ventricular ejection fraction (LVEF)  $\leq 50\%$  (OR: 3.9;  $p = .009$ ), Type D personality (OR: 8.9,  $p = .0001$ ), and age  $\leq 55$  years (OR: 2.6;  $p = .05$ ) as independent predictors of cardiac events. The presence of two or three of these risk factors occurring simultaneously increased the rate of poor outcome 4-fold ( $p = .0001$ ). Emotionally stressed, younger patients that have a decreased LVEF have an increased risk of cardiac events. Furthermore, when these risk factors coexist, a non-response to treatment may occur. Emotionally stressed younger patients with CHD represent high-risk groups that deserve more intense study.

Van der Broek, Martens, Nyklicek, Van der Voort, and Pedersen (2007) studied the emotional influence of not having a partner and its combined effects with Type D personality on anxiety and depression. The subjects were patients ( $n = 554$ ) that were hospitalized for acute

myocardial infarction (AMI) or an implantation procedure of a cardioverter defibrillator. These patients completed a 14-item Type D Scale (DS14) during their hospitalization and the State Trait Anxiety Inventory (STAI) and BDI at a 2-month follow up. The results showed that, stratifying by personality and partner status, Type D patients that did not have a partner had a higher risk of both anxiety (OR = 8.27, 95% CI, 2.50-27.31) and depressive symptoms (OR = 6.74, 95% CI, 2.19-20.76) followed by Type D patients that did have a partner (OR = 2.04, 95% CI, 1.05-3.96 and OR = 3.03, 95% CI, 1.46-6.31, respectively). It would seem that lack of a partner further exacerbates the risk of symptoms of anxiety and depression in the already identified Type D patient.

Pedersen, Denollet, Van Gestel, Serruys, and Van Domburg (2008) decided that the single factor approach to studying their influence on health outcomes in cardiac patients might not be giving the full picture. The impact of clustering was examined (Type D personality and anxiety) on depressive symptoms 12 months following percutaneous coronary intervention (PCI). Patients that had been treated with PCI ( $n = 416$ ) with drug eluting stents, were asked to complete the Type D Scale and the Hospital Anxiety and Depression Scale anxiety subscale at baseline, and the depression subscale at 12 months follow up. The results showed that of all these patients, 27% experienced depressive symptoms at 12 months. Depression could not, however, be attributed to cardiac events that had occurred during follow up ( $p = .76$ ). The prevalence of depressive symptoms at 12 months was the highest in patients that had clustering (64%), followed by the single risk factor of Type D (45%), and anxiety (38%). The lowest prevalence was in the “no risk factor” group (13%;  $p = .001$ ). The “single risk factors” Type D personality (OR = 5.82, 95% CI, 2.93-11.56) and anxiety (OR = 4.36, 95% CI, 2.23-8.55) and their co occurrence (OR = 12.38, 95% CI, 6.11-25.09) remained independent significant predictors of depressive symptoms at 12 months compared with the “no risk factor” group. Patients that have a co-occurrence of anxiety and Type D personality, which are two risk factors that are independently associated with

adverse prognosis, had a considerably higher risk of depressive symptoms 12 months post PCI compared with patients with no or just one risk factor.

The literature would suggest that depression and Type D personality have both been associated with worse outcome and prognosis for cardiac patients, but De Jonge et al. (2007), were concerned that the link between depression and cardiac prognosis could be confounded by somatic health. The association between depressive disorder and Type D personality was studied based on an individual's baseline somatic health. Post-MI patients ( $n = 1,205$ ) from the Myocardial Infarction and Depression Intervention Trial study were assessed according to the ICD-10 criteria with the Composite International Diagnostic Interview during the post-MI year and Type D patients with the DS14 at one year follow up. Somatic health was determined by left ventricular ejection fraction (LVEF), Charlson Comorbidity Index, previous MI, and CABG or percutaneous transluminal coronary angiography (PTCA). The results showed that the prevalence rates were 17.1% for post-MI depression and 18.7% for Type D. After controlling for confounders, post-MI depression was associated with lower baseline LVEF (OR = 3.17, 95% CI, 2.28-4.41) and greater co morbidity (OR = 1.46, 95% CI, 1.02-2.09), whereas Type D personality was not (LVEF: OR = 1.31, 95% CI, 0.93-1.87) and (comorbidity: OR = 0.92, 95% CI, 0.63-1.35). It would appear that post-MI depression is more related to somatic health and symptoms than Type D personality at 12-month follow up.

Literature exists primarily in favor of the Type D personality as it relates to increasing the risk for developing depressive symptoms (Denollet et al., 1996, 2000, 2006; Pedersen et al., 2008). Interestingly, Denollet was an integral part of most of the studies that were for or against the assumptions that were exerted about Type D personality. Pedersen et al. (2008) suggested that clustering of risk factors may have even greater impact than single risk factors and De Jonge et al. (2007) asserted that somatic health played a more integral part in depressive symptoms than Type D personality. It would appear that more research is needed, but that Type D personality is a

concept that will be shown to have its part in the development of depressive symptoms for cardiac patients.

## **Gender**

Gender differences in depression have been documented for several years and have only recently been thought to be important in the treatment selection for the depressed patient. Some general explanations for the male-to-female sex ratio in the prevalence of depression might include: psychological, neuro-chemical, anatomic, hormonal, genetic, and personality factors. Hormonal status may play an important role in addition to effects from the menstrual cycle, pregnancy, peri-menopause, and menopause (Grigoriadis & Robinson, 2007).

Romans, Tyas, Cohen, and Silverstone (2007) collected data from the Canadian Community Health Survey 1.2 for gender analysis of individual symptoms and overall rates of depression in the preceding 12 months. MDD was assessed by using the Composite International Diagnostic Interview. This represented a national, cross-sectional survey. Interestingly, the female to male ratio of MDD prevalence was 1.64:1 ( $n = 1766$ ). Women reported statistically more depressive symptoms than men ( $p < .001$ ).

Zinn-Souza et al. (2008) conducted a study to assess factors associated with symptoms of depression within a high school-aged sample (14-18 years). Seven hundred and twenty four students answered questionnaires based on life and health conditions. Factors associated to depressive disorders were analyzed by using multiple logistic regression. The results showed that over all, the prevalence rate of depression was 7.5%. These rates were divided into gender and found that 39 (10.3%) were females and 15 (4.3%) were males. Multiple logistic regression analysis showed that the factors associated with depressive disorders were being female (OR = 2.45), poor self-perception of health (OR = 5.78), and alcohol consumption (OR = 2.35).

Ryan et al. (2008) studied the relationship between gender and depression when



antidepressant medications were used. Method-adjusted Cox proportional hazards models were used to determine the association between depression and/or antidepressant use and 4-year survival in 7,363 community-dwelling elderly people. MDD was evaluated using DSM-IV criteria. Depressive symptoms were assessed using the CES-D. The results showed that mortality was the greatest in depressed men using antidepressants with increasing severity correlating to a higher hazard risk. Women with severe depression in the absence of treatment, on the other hand, had the strongest association with mortality. The conclusions show that the association between mortality and depression is gender-dependent and can vary in accordance with antidepressant use and symptom-load.

Danielsson and Johansson (2005) conducted a qualitative study to examine and explore depression from a gender perspective. They worked to capture depressed men's and women's formulations of their experiences and their particular understanding of the situation. The study took place in a healthcare center in northern Sweden. The subjects consisted of 18 patients that had been diagnosed with depression and had been treated for the depression for at least 6 months. The patients were of different ages and social status. These patients were interviewed in depth. Questions were posed and were based on the themes of Malterud's key questions. The focus was especially on how the informants conveyed their experiences. The results showed that the experience of depression was similar for men and women, but the outward manifestations differed by gender as well as socioeconomic status. Men spoke more easily about physical distress than emotions. Women spoke more readily about emotional distress, shame, and guilt, while physical symptoms tended to revolve around the stomach. Men dealt with insecurity by aggrandizing their previous competence. Women dealt with insecurity by self-effacement. The authors felt that clinicians must listen carefully to not only the words spoken but to the avoided words. This could help to counteract the normative gender patterns that highlight depression of women and tend to conceal that of men.

Gender and depression is also a highly studied issue as it relates to the cardiac patient. Westin, Carlsson, Erhardt, Cantor-Graae, and McNeil (1999) conducted a study in Sweden from 1989-1992 to evaluate the differences in QOL in male and female patients after AMI, CABG, and percutaneous transluminal coronary angioplasty (PTCA). Psychological and somatic dimensions of QOL were assessed by self-report questionnaires in these patients at one month ( $n = 376$ ) and one year ( $n = 349$ ) following the cardiac event. Normal controls were used for comparison ( $n = 88$ ). The results showed that female patients had a poorer QOL after 1 month (in depression, feeling of arrhythmia, anxiety, general health, self-esteem, and experience of sex life), and after 1 year as compared to their male counterparts.

Heo, Moser, and Widener (2007) also studied the health-related quality of life (HRQOL) in cardiac patients. The aim of the study was to investigate whether the gender might affect HRQOL in patients with heart failure (HF). Data were collected from 51 men and 47 women. The data were analyzed using regression analyses. The authors found no gender differences in the dynamic relationships among the variables in physical or emotional symptom status, or HRQOL, but there were differences found in the relationships between other dynamic variables. Using bivariate analyses, physical and emotional symptoms status was found to be related to HRQOL in both men and women. However, in women, HRQOL was related to physical symptom status, while in men depression affected HRQOL in multivariate analyses ( $p < .001$ ,  $r(2) = .27$ ;  $p < .001$ ,  $r(2) = .40$ , respectively). Functional status as measured by the NYHA Class mediated the effects of anxiety and depression on HRQOL only in women.

Verhagen et al. (2008) went a step further to examine if familiarity of MDD could contribute to observed gender differences in comorbidity. Familial (f-MDD) and non-familial MDD (n-MDD) cases were studied. This population sample was assessed for co morbid dysthymia, anxiety disorders, and alcohol related disorders. The Composite International Diagnostic Interview (CIDI) was used to assess this. Logistic regression analyses showed that

women with f-MDD reported a significantly higher amount of comorbid dysthymia and generalized anxiety disorder (GAD) than their male counterparts. Women that had n-MDD reported significantly more comorbid simple phobias and agoraphobia than their male counterparts. The authors concluded that models that explain comorbidity patterns of MDD do differ by gender.

Faller et al. (2007) went a step further to see if the influence of depression on health outcomes in heart failure patients could be affected by gender differences. At entrance to the study, depression was measured using a self-reported PHQ ( $n = 231$ ). The median follow-up time was 986 days. The results showed that suspected major depression was identified in 13% of patients. Minor depression was identified in 17% of patients. Interestingly, no differences between the sexes were discovered. Major, but not minor, depression was associated with increased mortality risk (HR = 3.3, 95% CI, 1.8-6.1,  $p = < .001$ ). The authors concluded that there is definitely a high prevalence of depression in the heart failure patient, but when testing the effect of interaction between depression and gender, statistical significance failed to be reached.

Naqvi et al. (2007) examined the influence of gender on the prevalence of ACS and the severity of depressive symptoms post-ACS. Nine hundred and forty four (716 men and 228 women) patients were surveyed using the Zung self-assessment questionnaire at discharge following unstable angina (UA) or ACS. Patients were asked to return the questionnaire by mail. Major depressive symptoms were diagnosed using a summed depressive symptoms (SDS) score  $> 50$ . Of these patients, 250 (35%) men, and 103 (45%) women had depressive symptoms ( $p = .005$ ). Depression was modeled by stepwise multivariable logistic regression using the following predictors: gender, age, hypertension, diabetes mellitus, history of smoking, hypercholesterolemia, peripheral vascular disease, prior stroke, prior MI, and prior percutaneous coronary intervention (PCI), or CABG. The significant predictor of depressive symptoms being present were female gender (OR = 1.64, 95% CI, 1.19-1.28), diabetes mellitus (OR = 1.42, CI

95%, 1.03-1.97), prior MI (OR = 1.56, 95% CI, 1.15-2.20), and smoking (OR = 1.41, 95% CI, 1.01-1.97). The variables that were associated with a higher severity of depressive symptoms were female gender, prior MI, smoking, and stroke. It was discovered that men who had prior MI, had higher SDS scores than men without prior MI (48.4 vs. 44.6, respectively,  $p = .001$ ). Interestingly, prior MI did not seem to affect SDS scores in women (49.1 for prior MI vs. 48.5 for no prior MI). Depressive symptoms were more severe in women with UA (SDS = 49.0) compared with women with AMI (SDS = 45), or men with AMI (SDS = 45.0,  $p = .004$ ) or UA (46.0,  $p = .007$ ). Although female gender is a significant independent predictor of depressive symptoms post-UA and MI, in women, a history of MI is associated with a higher frequency of depressive symptoms than in men.

The current evidence suggests that depression appears to cause a higher increase in coronary artery disease (CAD) in women, and that women CAD patients do experience greater levels of depression than men (Moller-Leimkuhler, 2007). It appears that whether one researches gender and depression from a non-specific view or from a disease-specific view (i.e., CHD), gender does seem to play a large role (Heo et al., 2007; Romans et al., 2007; Zinn-Souza et al., 2008). Very few studies show any indication of non-significance (Faller et al., 2007). Furthermore, when familial MDD is factored in, co morbid dysthymia is even greater in women than in men (Verhagen et al., 2008).

### **NYHA Class/Disability**

In 1928, the NYHA published a classification system for cardiac patients based on clinical severity and prognosis. These classifications have been updated in seven subsequent editions. The ninth, and most current, edition was revised by the Criteria Committee of the American Heart Association, New York City Affiliate (see Table 1). The class is defined by an

individual's functional capacity. The classes range from I to IV, with IV representing the lowest functional capacity. There is also an objective assessment component that is addressed by the

Table 1

*Nomenclature and Criteria for Diagnosis of Diseases of the Heart and Great Vessels*

Functional capacity	Objective assessment
Class I. Patients with cardiac disease but without resulting limitation of physical activity. Ordinary physical activity does not cause undue fatigue, palpitation, dyspnea, or anginal pain.	No objective evidence of cardiovascular disease.
Class II. Patients with cardiac disease resulting in slight limitation of physical activity. They are comfortable at rest. Ordinary physical activity results in fatigue, palpitation, dyspnea, or anginal pain.	Objective evidence of minimal cardiovascular disease.
Class III. Patients with cardiac disease resulting in slight limitation of physical activity. They are comfortable at rest. Less than ordinary activity causes fatigue, palpitation, dyspnea, or anginal pain.	Objective evidence of moderately severe cardiovascular disease.
Class IV. Patients with cardiac disease resulting in inability to carry on any physical activity without discomfort. Symptoms of heart failure or the anginal syndrome may be present even at rest. If any physical activity is undertaken, discomfort is increased.	Objective evidence of severe cardiovascular disease.

*Adapted from The Criteria Committee of the New York Heart Association, 1994.*

health care provider-based measurements taken such as electrocardiograms, stress tests, x-rays, echocardiograms, and radiological images (The Criteria Committee of the New York Heart Association, 1994).

This classification is used most often in patients who have CHF. Depression is common in this group of patients and is associated with poor QOL. Much research has been devoted to understanding how NYHA Class affects depressive symptoms in these patients. Moser, Doering,

and Chung (2005) conducted a study to look at the multiple risk factors that increased re-hospitalizations for patients being discharged with heart failure. Two hundred and two patients were evaluated for the following potentially modifiable risk factors: functional status, if patient lived alone, presence of depression, anxiety, poor QOL, symptom status and adherence to prescribed medications, symptom monitoring, and low-sodium diet. A good portion of these patients were severely functionally impaired (70% NYHA class III/IV). Of these patients, 69% were depressed.

Scherer et al. (2007) conducted a study to analyze depression and/or anxiety in primary care patients that had heart failure. They also tried to identify protective factors to help resolve these psychosocial distresses. The study was a longitudinal observation study in primary care practices in lower Saxony, Germany ( $n = 291$ ). Depression and anxiety were assessed using the Hospital Anxiety and Depression Scale (HADS). Severity of heart failure was assessed using the NYHA classification system. The results showed that 26 (32.5%) of those patients that were distressed at baseline had a normal HADS score 9 months later. The rest of these patients remained distressed. Using logistic regression, baseline distress (OR 5.51%; 95% CI = 2.56 - 11.62), emotional problems (OR 1.08; 95% CI = 1.00 - 1.17), social support (OR 0.54; 95% CI = .35 - .83), and NYHA classification (OR 1.70; 95% CI = 1.05 - 2.77) independently predicted distress at follow up. High social support aided in the resolution of depression and anxiety and partnership and low levels of emotional problems protected patients that had started the study in a good emotional state. This study suggests that high NYHA class and emotional problems may contribute to depression and anxiety.

Freedland et al. (2003) conducted a study to investigate the prevalence of depression in 682 hospitalized heart failure patients. The study also identified demographic, psychosocial, medical, and methodological factors that might affect prevalence. A modified version of the DSM-IV was administered to these patients to identify major and minor depression. Six hundred

and thirteen of these patients also completed the BDI. The medical, demographic, and social data were obtained through hospital chart review, patient interview, and echocardiography. In this particular sample, 20% of the patients met DSM-IV criteria for current MDD, and 51% scored higher than the cutoff for depression using the BDI ( $\geq 10$ ). Interestingly, the prevalence of MDD differed significantly between the functional severity of the heart failure (NYHA class), gender, age, employment status, past history of MDD, and dependence in activities of daily living (ADL). The prevalence of depression ranged from as low as 8% in patients that had a NYHA class of I, to as high as 40% in patients that had NYHA class of IV.

Pihl, Jacobsson, Fridlund, Stromberg, and Martensson (2005) wanted to determine if older patients suffering with heart failure and their spouses experienced similar levels of depression. Data were collected from 47 couples. The Zung Self-rating Depression Scale (SDS) and the Short Form 36 (SF-36) were used. The results showed that patients suffering from heart failure, along with their spouses, did not differ significantly in their mental health. Physical symptoms primarily dominated the experience of the depressive experience of the patient, which, in turn, affected the spouse accordingly. The patient's depression was positively related to NYHA class, while the spouse's depression was related more with the higher age of the patient.

Jiang et al. (2001) conducted a study to determine the prevalence of depression in hospitalized CHF patients that were in the NYHA Class II or greater. The patients were all 18 years of age and older, had an ejection fraction of 35% or less, and were admitted between March 1, 1997, and June 30, 1998 to a cardiac care unit. Patients were given the BDI to assess depression. If a patient scored 10 or greater, they underwent a modified National Institute of Mental Health Diagnostic Interview Schedule to assess for MDD. All cause mortality and readmission rates were assessed. Of 374 patients screened, 35.3% had a BDI score of 10 or greater, and 13.9% had MDD.

Gott et al. (2006) studied the factors predictive of QOL amongst older people that had heart failure. Five hundred and forty two patients aged over 60 years with heart failure were recruited from community general practice surgeries. Patients completed a postal questionnaire that included disease-specific questions (Kansas City Cardiomyopathy Questionnaire), a generic QOL questionnaire (SF-36), and provided sociodemographic information. A multiple linear regression analysis identified the following factors as predictors of decreased QOL: NYHA functional class III or IV, evidence of depression, female, lower socioeconomic group, and two or more medical comorbidities.

Westlake, Dracup, Fonarow, and Hamilton (2005) conducted a study to describe the prevalence of depression in patients with heart failure and the differences between patients with minimal versus mild to severe depression. The data were collected from 200 patients having symptoms of heart failure that resulted from systolic dysfunction. These patients were, on average, 57.0 (+/- 12.1) years old, male (168, 84%), and in the NYHA functional class II or III ( $n = 140$ , 70.0%), with a mean ejection fraction (EF) of 25.5 +/- 6.4%. Minimal depression was described by 105 (52.5%) patients, mild depression was described by 62 (31%) patients, moderate depression was described by 30 (15%) patients, and severe depression was described by 3 (1.5%) patients. The significant differences between patients with minimal depression compared to mild to severe depression, were NYHA class ( $\chi^2 = 14.05$ ,  $p = .003$ ), maximal oxygen uptake ( $t = 2.62$ ,  $p = .010$ ), 6 minute walk distance ( $t = 4.22$ ,  $p < .001$ ), beta blocker therapy ( $\chi^2 = 15.21$ ,  $p < .001$ ), perceived control ( $t = 7.93$ ,  $p < .001$ ), and neuroticism ( $t = -8.85$ ,  $p < .001$ ).



## **The Prevalence of Depression in the Cardiac Patient**

The prevalence of depression in the cardiac patient can range anywhere from 15-65% (Amin et al., 2005; Feinstein et al., 2006; Hermann-Lingen, 2001; Kaptein et al., 2005). Many studies exist that include statistics specifying the prevalence of depression in the patient with CHD (Carney et al., 1996; Goldstein, 2006; Hermann-Lingen, 2001; Lane et al., 1999; Thornton, 2001). For instance, evidence suggests that depression experienced after an MI is not transient. At 3 months follow up, 77% of patients who met the criteria for major depression still showed evidence of depression (Lane et al., 1999). Data from epidemiological surveys suggest that the typical cardiologist doing rounds on a 25-bed unit will see four patients with major depression and five with a minor form of depression (Lesperance & Frasure-Smith, 2000). The occurrence of depression before and after CABG surgery was 53% at one month after surgery and 47% at one year. In patients that were not depressed before CABG surgery, 13% were depressed at one month and 9% were depressed at one year (Lane et al., 1999). Of 430 patients with unstable angina, 41% were identified as depressed (Goldstein, 2006).

### **Following Hospitalization for MI**

Frasure-Smith (2000) conducted a study that initially interviewed 222 MI patients from 5 to 15 days following their admission to the hospital. Forty-seven percent of these patients met criteria for major depression either by the DSM-III-R or BDI scores. Of those who met criteria at 6 months follow up, 5% of these patients had died, and half of them had major depression; this suggests a relative risk associated with the depression of 4.29 (95% CI, 3.14-5.44) after controlling for other clinical variables (Killip class, previous MI, and prescription of Warfarin at discharge).

Murphy et al. (2008) conducted a study to examine different trajectories of anxiety and

depression after an AMI in the 12 months following their cardiac event. A sample of 226 women was interviewed using the Hospital Anxiety and Depression Scale (HADS) on four separate occasions. These women had experienced either an AMI or CABG. Growth curve and growth mixture modeling was used in order to identify trajectories of change, and univariate tests were used to discover predictors of each trajectory. In general, most of these women began with relatively low levels of anxiety and/or depression. These conditions improved over the 12-month period of follow up (84% of women showed this trajectory for anxiety and 89% showed this trajectory for depression). Interestingly, a smaller group began with relatively high levels of anxiety and/or depression that actually got worse over time (16% for anxiety and 11% for depression). The patients in the high level group were more likely to report high levels of loneliness, to not have English as a first language, to perceive their cardiac disease as more severe (anxiety group only) and have diabetes (depression group only).

Kaptein et al. (2006) conducted a study to classify MI patients according to their specific course of depressive symptoms and to then evaluate their cardiac prognosis. In the process, much was discovered about the prevalence of depression in these patients. Data were derived from the Depression after Myocardial Infarction (DepreMI) study. This study was a naturalistic follow-up study of patients that were admitted with an MI to four different hospitals in the Netherlands during September 1997 and September 2000 ( $N = 475$ ). The BDI was administered to patients at 3, 6, and 12 months post-MI and was then analyzed. From the analyses, it was discovered that the prevalence of significant depressive symptoms ranged anywhere from 22.7% to 25.5% during the post-MI year. Five distinct courses were identified: no depressive symptoms (56.4%), mild depressive symptoms (25.7%), moderate and increasing depressive symptoms (9.3%), significant but decreasing symptoms (4.6%), and significant and increasing symptoms (4.0%). Incidentally, patients in the last class identified, had a statistically significantly higher risk for a new

cardiovascular event compared to subjects without depressive symptoms (hazard ratio [HR] = 2.73;  $p = .01$ ).

Lehto et al. (2000) set out to study the prevalence of depression at least 6 months following a CHD event. These events could include MI, bypass grafting, coronary angioplasty, and myocardial ischemia without infarction. The study was titled The EUROASPIRE Study (European Action on Secondary Prevention through Intervention to Reduce Events). This was a multi-center study conducted in nine European countries. The subjects consisted of 414 patients (284 males and 130 females) who were younger than 71 years (mean age for men was 60.9 years and for women was 63.6 years). These subjects were interviewed and examined and had their medical charts reviewed. The NYHA Class was also assessed. Among these four diagnostic categories, one-sixth (14-19%) of the patients suffered from depression. The depression was highly associated with smoking (OR 1.7, 95% CI-1.2; 2.4) and poor NYHA class (OR 1.9, 95% CI 1.4; 2.6).

The Cardiovascular Health Study followed 5,888 subjects over 65 years of age that had no known current or prior cardiovascular disease (CVD). Four thousand four hundred and ninety-three subjects were free of CVD at baseline as assessed by the CES-D. These 4,493 participants were followed for 6 years for the development of CVD and mortality. The cumulative mean depression score was assessed for each participant up to the time of a cardiac event (maximum 6 year follow up). With every five-unit increase in mean depression score for the development of CVD was 1.15 ( $p = .006$ ); the ratio for all cause mortality was 1.29 ( $p < .0001$ ). Among participants with the highest cumulative mean depression scores, the risk of CVD increased by 40% and the risk of death increased by 60% compared with those who had the lowest mean scores (Ariyo et al., 2000).

Lauzon et al. (2003) conducted a study that involved five tertiary care and five community hospitals. These patients had been admitted for MI within two to three days of

admission. The patients were then recruited for the study by trained nurse interviewers. Information was collected from patient medical charts and from results obtained from the BDI. The BDI was administered and completed during the patients hospital stay and then mailed questionnaires were sent out at 30 days, 6 months, and 1 year after discharge. Of the 587 subjects, 550 (94%) actually completed the BDI at baseline and of those 94%, 191 (35%) had a score of 10 or more that would indicate at least mild depression. It was discovered that the rates of depression did not vary significantly over the follow-up period. These results were similar among patients that were admitted to tertiary care or community hospitals.

Krzyzkowiak (2007) conducted a study to assess depressive symptoms following an MI. Structured interviews including, Mini International Neuropsychiatric Interview, BDI, Social Readjustment Rating Scale, and Recent Life Changes Scale, were used in the analysis of 102 patients hospitalized for MI. The findings showed that clinical (major) depression was diagnosed in 10.8% of these patients following an MI. However, depressive symptoms (> 10 points on the BDI) were detected in 40% of these patients.

The research seems to indicate that depression is generally quite widespread among cardiac patients following an MI (Frasure-Smith, 2000; Lehto et al., 2000). Murphy et al. (2008) found that individuals that began with high levels of either depression and/or anxiety, actually got worse over time. Kaptein et al. (2006) further went on to develop a system to identify different courses of depression following an MI with the last class identified (significant and increasing symptoms of depression), to have a statistically significantly higher risk for new cardiovascular events.

### **Following Hospitalization for CABG**

Depression following CABG surgery is common. Depression has been found to predict slower recovery, increased prevalence of further depressive symptoms, and increased risk for developing further cardiac events (Pedersen et al., 2008).

At Duke University, 817 patients were about to undergo CABG surgery between May 1989 and May 2001. Patients completed the CES-D scale prior to surgery, 6 months following surgery, and then were followed up on for a maximum of 12 years. Of these 817 patients, there were 122 deaths (15%) in a mean follow up of 5.2 years. Three hundred and ten patients (38%) met the criterion for depression (CES-D = 16), 213 (26%) for mild depression (CES-D = 16-26), and 97 (12%) for moderate to severe depression (CES-D = 27). When controlling for age, sex, number of grafts, diabetes, smoking, and left ventricular ejection fraction, survival analysis showed that patients with moderate to severe depression at baseline (adjusted HR 2.4, CI 1.4-4.0);  $p = .001$ ) and mild or moderate to severe depression that persisted from baseline to 6 months (adjusted HR 2.2, (1.2-4.2);  $p = .015$ ) had higher rates of death than did those with no depression (Blumenthal et al., 2003).

Connerney, Shapiro, McLaughlin, Bagiella, and Sloan (2001) assessed the effects of depression on specific outcomes following coronary artery bypass graft (CABG) surgery. Just prior to discharge, depression was assessed using a structured psychiatric interview and a questionnaire, the BDI. During the first year after surgery, 201 men and 102 women were followed. Out of the 303 patients surveyed, 63 patients (20%) met the modified diagnostic statistical manual IV criteria for MDD.

Rafanelli, Roncuzzi, and Milaneschi (2006) conducted a study to assess clinical and subclinical distress in a sample of patients who had undergone CABG surgery at 1 month and then at a 6-8 year follow up. Although the main findings of the study addressed the relationship between psychological variables and coronary events, it was discovered that of the 47 patients

that were interviewed using both the Diagnostic and Statistical Manual (DSM) and the new Diagnostic Criteria for Psychosomatic Research (DCPR), 36% of these patients were given a psychiatric diagnosis, and practically half of the sample met the criteria for a DCPR cluster.

What seems to be of even more interest is the connection that appears to exist between depression after CABG surgery and gender. It appears as though women tend to fare less favorably after CABG surgery, and a great deal of research exists to support such claims (Mitchell et al., 2005).

Burker et al. (1995) conducted a longitudinal study in order to determine the prevalence of depression in male and female patients about to undergo cardiac surgery. The study also examined what factors were associated with depression before and after surgery. One day before surgery (T1), and one day before hospital discharge (T2), 141 patients completed a battery of psychometric tests. These tests included the CES-D, the State-Trait Anxiety Inventory (STAI), and the Perceived Social Support Scale (PSSS). Data were also collected on 13 different physiological measures. Of these patients, 47% were considered to be depressed as defined by a score of 16 or above on the CES-D at T1. Interestingly, scores increased significantly from T1 ( $M = 15$ ) to T2 ( $M = 20$ ), with 61% of patients classified as depressed at T2. The factors associated with depression at T1 were female gender, higher state anxiety, and less social support. The patients depressed at T2 were characterized by higher scores on the STAI at T2 and higher scores on the CES-D at T1.

Vaccarino et al. (2003) conducted a study to explore whether female gender was associated with poorer outcomes following CABG surgery. The first 4 to 8 weeks following CABG surgery are generally the most vulnerable of the entire recovery period. This study followed 1,113 patients (804 men and 309 women) undergoing their first CABG between February 1999 and February 2001. Patients were interviewed at baseline, pre surgery, and between 6 to 8 weeks post surgery. The clinical data were extracted from medical records. The

findings showed that compared to men, women tended to be older and more often had unstable angina and CHF, lower physical function (PF), and greater depressive symptoms pre surgery. At 6 to 8 weeks following CABG surgery, following adjustment for baseline characteristics, the rate of hospital readmission was 20.5% in women and 11.0% in men ( $p = .005$ ). The mean number of physical symptoms and side effects was 2.5 in women and 2 in men ( $p = .0009$ ). The PF remained unchanged in men (an increase in score of 0.3 points, 95% CI, -1.1-1.9), and depressive symptoms improved (a decrease of 0.2 depressive symptoms, 95% CI, -0.4- -0.04). Women showed, on average, a 13-point decline in PF (95% CI, -15.8 - -10.4), and an increase of 0.5 in depressive symptoms (95% CI, 0.1-0.9). It seems that women have a more difficult recovery compared to men, which is not explained by illness severity, pre-surgery health status, or other patient characteristics.

Mitchell et al. (2005) did conduct some research that disputed the above results, however. Setting out to test the hypothesis that women fare worse than men after CABG surgery, this study revisited the sex differences in depression during recovery after this surgery. The study followed 137 patients (72 men and 65 women) that were about to undergo an elective first CABG surgery between July 2003 and April 2004. An interview was conducted  $\leq 28$  days before surgery and then again somewhere between 6 and 12 weeks following surgery, utilizing a structured diagnostic interview, by a clinician, for MDD and the BDI. The data were retrieved from patient charts. In both groups, the prevalence of MDD was 28.2%, possibly due to the stress of preparing for the surgery, but this number decreased to 16.4% after surgery ( $p = .038$ ). In comparison, women had significantly more depression than men pre-CABG, with a mean BDI of 12.5 (95% CI, 10.6-14.4) versus 8.0 (95% CI, 6.3-9.8) for men ( $p = .0001$ ), but not post surgery. Interestingly, there was a significant sex-by-time interaction with depressive symptoms in women that showed an almost 6-fold improvement greater than in men. The BDI change scores were 4.1 (95% CI, 2.0- 6.1) for women versus 0.7 (95% CI, 1.0-2.5) for men ( $p = .008$ ). The interaction

even remained significant after adjusting for such baseline characteristics as education, social support, and operative risk. This study shows that women benefited from CABG as much or even more than men in terms of their mental health.

A great deal of research points in the direction of increased depression following CABG surgery (Connerney et al., 2001; Rafanelli et al., 2006). There is also a trend in the research that points toward gender playing a part in the recovery following CABG (Vaccarino et al., 2003). Mitchell et al. (2005) disputes this claim as far as long-term outcome is concerned, but does report that women initially (pre surgery), do have higher levels of depression than men. The findings do suggest that there is something to the notion of gender and recovery from CABG.

### **Following Hospitalization for CHF**

Depression is common in CHF patients and is associated with poor QOL. Much research has been devoted to understanding how NYHA Class affects depressive symptoms in these patients (Freedland et al., 2003; Moser et al., 2005; Scherer et al., 2007). Furthermore, NYHA Class is one of the greatest predictors toward developing depression in patients who have CHF (Gott et al., 2006).

Moser et al. (2005) conducted a study to look at the multiple risk factors that increased rehospitalizations for patients being discharged with heart failure. Two hundred and two patients were evaluated for the following potentially modifiable risk factors: functional status, if patient lived alone, presence of depression, anxiety, poor QOL, symptom status and adherence to prescribed medications, symptom monitoring, and low-sodium diet. A good portion of these patients were severely functionally impaired (70% NYHA class III/IV). Of these particular patients, 69% of them were depressed.

Scherer et al. (2007) conducted a study to analyze depression and/or anxiety in primary care patients that had heart failure and also tried to identify protective factors to help resolve these



psychosocial distresses. The study was a longitudinal observation study in primary care practices in lower Saxony, Germany ( $n = 291$ ). Depression and anxiety was assessed using the Hospital Anxiety and Depression Scale (HADS). Severity of heart failure was assessed using the NYHA classification system. The results showed that 26 (32.5%) of those patients that were distressed at baseline had a normal HADS score 9 months later. The rest of these patients remained distressed. Using logistic regression, baseline distress (OR 5.51%; 95% CI = 2.56 - 11.62), emotional problems (OR 1.08; 95% CI = 1.00 - 1.17), social support (OR 0.54; 95% CI = .35 - .83), and NYHA classification (OR 1.70; 95% CI = 1.05 - 2.77) independently predicted distress at follow up. High social support aided in the resolution of depression and anxiety and partnership and low levels of emotional problems protected patients that had started the study in a good emotional state. This study suggests that high NYHA class and emotional problems may contribute to depression and anxiety.

Freedland et al. (2003) conducted a study to report the prevalence of depression in 682 hospitalized heart failure patients. The study also identified demographic, psychosocial, medical, and methodological factors that might affect prevalence. A modified version of the DSM-IV was administered to these patients to identify major and minor depression. Six hundred and thirteen of these patients also completed the BDI. The medical, demographic, and social data were obtained through hospital chart review, patient interview, and echocardiography. In this particular sample, 20% of the patients met DSM-IV criteria for current MDD, and 51% scored higher than the cutoff for depression using the BDI ( $> \text{or} = 10$ ). Interestingly, the prevalence of MDD differed significantly between the functional severity of the heart failure (NYHA Class), gender, age, employment status, past history of MDD, and dependence in activities of daily living (ADL). The prevalence of depression ranged from as low as 8% in patients that had a NYHA class of I, to as high as 40% in patients that had NYHA class of IV.

Pihl et al. (2005) wanted to determine if older patients suffering with heart failure and

their spouses experienced similar levels of depression. Data were collected from 47 couples. The Zung Self-rating Depression Scale (SDS) and the Short Form 36 (SF-36) were used. The results showed that patients suffering from heart failure and their spouses did not differ significantly in their mental health. Physical symptoms primarily dominated the experience of the depressive experience of the patient, which, in turn, affected the spouse accordingly. The patient's depression was positively related to NYHA Class, while the spouse's depression was related more with the higher age of the patient.

Jiang et al. (2001) conducted a study to determine the prevalence of depression in hospitalized CHF patients that were in the NYHA Class II or greater. The patients were all 18 years of age and older, had an ejection fraction of 35% or less, and were admitted between March 1, 1997 and June 30, 1998 to a cardiac care unit. Patients were given the BDI to assess depression. If a patient scored 10 or greater, they underwent a modified National Institute of Mental Health Diagnostic Interview Schedule to assess for MDD. All cause mortality and readmission rates were assessed. Of 374 patients screened, 35.3% had a BDI score of 10 or greater, and 13.9% had MDD.

Gott et al. (2006) studied the factors predictive of QOL amongst older people that had heart failure. Five hundred and forty two patients aged > 60 years with heart failure were recruited from community general practice surgeries. Patients completed a postal questionnaire that included disease-specific questions (Kansas City Cardiomyopathy Questionnaire), a generic QOL questionnaire (SF-36), and provided sociodemographic information. A multiple linear regression analysis identified the following factors as predictors of decreased QOL: NYHA functional class III or IV, evidence of depression, female, lower socioeconomic group, and two or more medical comorbidities.

Westlake et al. (2005) conducted a study to describe the prevalence of depression in patients with heart failure and the differences between patients with minimal versus mild to

severe depression. The data were collected from 200 patients having symptoms of heart failure that resulted from systolic dysfunction. These patients were, on average, 57.0 (+/- 12.1) years old, male 168 (84%), and in the NYHA functional class II or III ( $n = 140$ , 70.0%), with a mean ejection fraction (EF) of 25.5 +/- 6.4%. Minimal depression was described by 105 (52.5%) patients, mild depression was described by 62 (31%) patients, moderate depression was described by 30 (15%) patients, and severe depression was described by 3 (1.5%) patients. The significant differences between patients with minimal depression compared to mild to severe depression, were NYHA class ( $\chi^2 = 14.05$ ,  $p = .003$ ), maximal oxygen uptake ( $t = 2.62$ ,  $p = .010$ ), 6 minute walk distance ( $t = 4.22$ ,  $p < .001$ ), beta-blocker therapy ( $\chi^2 = 15.21$ ,  $p < .001$ ), perceived control ( $t = 7.93$ ,  $p < .001$ ), and neuroticism ( $t = -8.85$ ,  $p < .001$ ).

The research seems to show that depression is extremely common in the patient with CHF (Freedland et al., 2003; Jiang et al., 2001; Moser et al., 2005). NYHA Class (I-IV) independently predicted distress at follow up after hospitalization (Sherer et al., 2007), and had the greatest bearing on developing depression for those with CHF (Gott et al., 2006).

### **The Effects of Untreated Depression**

Major depression is a debilitating comorbid disorder that can seriously complicate recovery and increases the risks of further cardiac morbidity and mortality (Carney et al., 1996). Depression has been associated with a worse prognosis and increased coronary events after MI whether depression was evident before the cardiac event or diagnosed after the cardiac event (Kemp et al., 2003). The more severe the depression is, the greater the risk of death following a cardiac event (Kemp et al., 2003). A great deal of research has been conducted to show the connection between depression and the increased risk of morbidity and mortality following a cardiac event. Nonadherence to a physician's medication treatment plan is increasingly recognized and can cause adverse cardiac outcomes (Gehi, Ali, Na, & Whooley, 2007). This

section will address the effects of untreated depression that include: increased risk of a coronary event, decreased feelings of wellbeing and QOL, decreased medication compliance, and decreased risk factor modification.

### **Increased Risk of a Coronary Event**

Barefoot and Schroll (1996) conducted a study spanning 27 years from 1964-1991 in Glostrup, Denmark. The study included 409 men and 321 women participants. Physical and psychological exams established each patient's baseline risk factor and disease status, and their level of depressive symptomatology. Initial MI was observed in 122 participants, and 290 deaths occurred during follow up, ending in 1991. A two standard deviation (*SD*) difference in depression score was associated with a relative risk of 1.71 ( $p = .005$ ) for MI and 1.59 ( $p = .001$ ) for deaths from all causes. These findings remain unchanged even after controlling for individual risk factors and sign of disease at baseline. This study concluded that high levels of depressive symptoms increase the risk of MI and mortality. The research suggests that depression is best viewed as a continuous variable that represents a chronic psychological characteristic rather than a discrete, episodic psychiatric condition. They found that depression associated with ischemic heart disease increased morbidity for both men and women with a 70% higher risk for re-infarction and a 60% higher mortality rate.

Parakh et al. (2008) was interested to see if the effects of depression on mortality in the short term following an MI could be persistent in the long term. The study tried to determine whether depression during hospitalization for MI, which could predict mortality at 4 months, could also predict mortality 8 years later. The study was prospective and observational and included 284 hospitalized patients with MI. Major depression and dysthymia were assessed using the Diagnostic and Statistical Manual of Mental Disorders, revised third edition. Depressive symptoms were assessed using the BDI. Mortality was determined by using the Social Security

Death Index. Any depression (major, dysthymia, and/or BDI score of  $\geq 10$ , was detected in 76 patients (26.8%). The 8 year mortality rate was 47.9% (136 deaths). Any depression at the time of the MI was not associated with mortality at 8 years in an adjusted hazard ratio (HR 1.25, 95% CI, 0.87-1.81,  $p = .22$ ).

Ariyo et al. (2000) went on further to investigate if depressive symptoms were a risk for CHD and mortality in an apparently healthy elderly sample. The study was prospective and included 5,888 elderly Americans that were enrolled in the Cardiovascular Health Study. Of these 5,888 participants, 4,493 were free of cardiovascular disease at baseline. The depression-free participants provided annual information regarding their depressive status as assessed by the CES-D. For 6 years, these participants were followed for the development of CHD and mortality. A cumulative mean depression score was assessed for each participant up until the time of a cardiac event. Using time-dependent, proportional-hazards models, the unadjusted hazard ratio that is associated with every five-unit increase in mean depression score for the development of CHD was 1.15 ( $P = .006$ ). Of the participants that had the highest cumulative mean depression scores, the risk of CHD increased by 40% and the risk of death by 60% compared with those who had the lowest mean scores.

### **Decreased Medication Compliance**

Medication compliance is a common problem in cardiovascular patients with depression (Goldstein, 2006; Lesperance & Frasure-Smith, 2000). Carney et al. (1996) conducted a study that showed that 69% of nondepressed subjects were adherent to their medication treatment plan, whereas only 45 % of the depressed group was compliant.

Gehi, Haas, Pipkin, and Whooley (2005) conducted a study to examine the association between current major depression, assessed by using the Diagnostic Interview Schedule, and self-reported medication adherence. The study was cross sectional and included 940 outpatients with

stable CHD. Of the participants, 204 (22%) had major depression. Of the 204 depressed patients, 28 (14%), reported not taking their medications as prescribed whereas 40 (5%) of 736 nondepressed patients reported the same (OR 2.8, 95% CI, 1.7-4.7;  $p < .001$ ). Two times as many depressed patients as nondepressed patients (18% vs. 9%) reported that they forgot to take their medications (OR, 2.4; 95% CI, 1.6-3.8;  $p = .001$ ). When asked about deciding to skip taking their medications, 9% of depressed and 4% of nondepressed reported doing so (OR, 2.2; 95% CI, 1.2-4.2;  $p = .01$ ). This relationship between depression and nonadherence continued to persist even after adjusting for certain confounding variables like age, ethnicity, education, social support, and cardiac disease severity (OR, 2.2; 95% CI, 1.2-3.9;  $p = .009$ ). This study appears to show an association between depression and medication nonadherence. Medication nonadherence may likely contribute to unfavorable cardiovascular outcomes.

Gehi et al. (2007) conducted a study to evaluate the risk of cardiovascular events associated with medication nonadherence. The participants were 1,015 outpatients with already established CHD. A single question was asked, “In the past month, how often did you take your medications as the doctor prescribed?” The nonadherence rate was defined as not taking prescribed medications 75% of the time or less. Of the 1,015 participants, 83 (8.2%) reported nonadherence to their medications, and 146 (14.4%) developed cardiovascular events. Furthermore, the self reported nonadherent patients were more likely than the adherent patients to develop subsequent cardiac events during 3.9 years of follow up (22.9% vs. 13.8%,  $p = .03$ ).

### **Decreased Feelings of Well-being/QOL**

Depression has been shown to lead to decreased feelings of overall well-being, or QOL (Feinstein et al., 2006; Hermann-Lingen, 2001). Participants with depressive symptoms are more likely to report mildly diminished QOL (Ruo et al., 2003).

Ruo et al. (2003) conducted a study that measured and compared the contributions of depressive symptoms to cardiac function, and health status of patients that had coronary artery disease. A cross-sectional study was conducted with 1,024 participants in the San Francisco Bay area from September 2000 to December 2002. QOL was one variable that was measured using the Seattle Angina Questionnaire. The results showed that of the 1,024 participants, 201 (20%) of these participants had depressive symptoms. Furthermore, participants with depressive symptoms were more likely to report mildly diminished QOL (67% vs. 31%;  $p < .001$ ).

Pedersen and Denollet (2004) conducted a study that focused on Type D personality. The study focused on whether patients with Type D personality were at increased risk for cardiovascular morbidity and mortality (OR ranging from 4.1-8.9,  $p < 0.0001$ ) independent of standard cardiac risk factors. These patients were also found to suffer from impaired QOL.

Heo et al. (2007) conducted a study to determine if there were gender differences in the effects of physical and emotional symptoms as they related to HRQOL. The subjects consisted of 51 men and 47 women with heart failure. The data were analyzed using regression analyses. No gender differences existed in physical or emotional symptom status, but there were differences in the dynamic relationships among the variables. In women, however, physical symptoms were related to HRQOL ( $r(2) = .27$   $p < .001$ ). In men, depression affected HRQOL ( $r(2) = .40$   $p < .001$ ). The results showed gender differences in the dynamic relationships among variables related to HRQOL.

The connection seems to exist that would suggest that if patients suffer from depressive symptoms, they are more likely to report decreased QOL (Ruo et al., 2003).

### **Physician Under Detection of Depression**

A diagnosis of depression is missed in 50% of all cases. Of those 50%, less than 10% of these cases have any kind of treatment instituted (Thornton, 2001). Amin et al. (2006) found that

hospitalized patients with moderate to severe depressive symptoms by PHQ were only recognized 24.5% of the time as evidenced by documentation in the patient chart. Despite its clinical importance and high remission rate, depression still goes under recognized and under treated in cardiac patients (Hermann-Lingen, 2001).

In a national survey, 796 cardiovascular physicians were polled and were found to be aware of the importance of depression in cardiovascular disease. This was evidenced by the fact that 84.8% of surveyed physicians believed that up to half of their patients with CHD have depression (Feinstein et al., 2006). Further questioning revealed that 71% of these physicians asked less than half of their patients with CHD about depression and 79% used no standard screening methods. Interestingly, only 55% of these physicians felt comfortable making a diagnosis of depression.

Several explanations have been identified to explain why physicians are missing the diagnosis of depression in their cardiac patients. Discriminating symptoms of depression from those related to other medical conditions, or comorbidities, can be difficult (Davies, Jackson, Potokar, & Nutt, 2004; Davies et al., 2004). Comorbidities not only make diagnosis difficult, but they also increase a patient's resistance to treatment (Rosenthal, 2003). Magruder-Habib et al. (1990) found that the prevalence and severity of a patient's comorbidities can also complicate diagnosis. They conducted a study at a medical clinic at a VA medical center in Durham, North Carolina. Eighth hundred and eighty male veterans were followed longitudinally by six board certified internists. These participants were screened for depression using three specific screening tools: (a) Diagnostic and Statistical Manual-III, (b) Self-Rating Depression Scale (SDS), and (c) Diagnostic Interview Schedule. Those patients who screened positive on at least two screenings, were randomly assigned to one of two groups: Group A, primary physician informed of screening results; or Group B, primary physician not informed of screening results. Physicians were blinded to the study hypothesis. One hundred patients met both screening criteria (12.7%). The results of



the study showed that feedback to physicians of screening tool scores of previously unrecognized depressed patients makes a significant difference in greater recognition (56.2% vs. 34.6%). This is especially true regarding patients with high somatic ( $p < .05$ ) or low psychologic symptoms of depression ( $p < .05$ ).

One possible explanation is that pain and depression share the same neurologic pathway (Trivedi, 2004). Somatic symptoms such as joint pain, limb pain, back pain, gastrointestinal problems, fatigue, psychomotor activity changes, appetite changes, diminished energy, and sleep disturbances, can overlap with those of medical illnesses (Goldstein, 2006; Trivedi, 2004). Somatic symptoms may be the only way some groups of people know how to express the presence of an emotional problem, especially if depression carries a cultural or group stigma. This is especially true in seniors, who may not know any other way to express these feelings (Rosenthal, 2003). In a general sense, the worse the physical pain and symptoms, the more severe the depression is (Trivedi, 2004).

Another potential barrier to recognition of depression in cardiac patients is the physician's preconceived notion that depressive symptoms may reflect a "reaction" to their cardiac event (Amin et al., 2005). It can oftentimes be difficult to distinguish transient psychological symptoms from those that represent major depressive illness (Davies et al., 2004).

Other reasons for low rates of detection include: characteristics of the patient, physician knowledge of depression, physician skill in recognizing and treating depression, a physicians negative attitude toward mentally ill patients, the quality and precision of diagnostic information available, and the time constraints imposed by managed care (Magruder-Habib et al., 1990; Rosenthal, 2003).

Physicians and patients need to become more aware of the broad spectrum of depressive symptoms in order to evaluate and treat depression more effectively (Trivedi, 2004). The diagnosis of depression depends on systematic patient evaluation by experienced clinicians

trained in the recognition of these disorders (Januzzi & Pasternak, 2002). Physicians also must learn to delegate information gathering to trained personnel, in the form of questionnaires and rating scales. This will allow the physician to assess the collected data and then focus on diagnostic issues (Rosenthal, 2003).

## CHAPTER III

### METHODOLOGY

This chapter provides an overview of the completed study, by reviewing (a) the research design, (b) sample, (c) instrumentation, (d) pilot testing, (e) statistical analyses used, and (f) data collection procedures.

#### **Research Design**

A one-group, cross sectional, research design was employed to measure the severity of depression in hospitalized cardiac patients. The data were gathered utilizing the PHQ-9, a self-report depression inventory (see Appendix A).

#### **Sample**

##### **Selection Criteria**

Utilizing convenience sampling, all patients who were admitted as inpatients in the critical care unit of a hospital in the intermountain west, who had a troponin blood test ordered, CABG, aortic valve replacement (AVR), mitral valve replacement, percutaneous transluminal coronary angioplasty (PTCA), stent, CHF, atrial fibrillation, unstable angina, ACS, pacemaker implantation, MI, or chest pain, were considered for inclusion in this study. Patients were excluded if they had altered levels of consciousness ( $n = 5$ ), deceased before hospital discharge, were under the care of hospice, or were non-English speaking ( $n = 21$ ).

This researcher approached and introduced herself to patients during their hospitalization that met the study criteria. The content of the study was explained and patient was asked if he/she would like to participate. If the patient agreed to participate, then informed consent was obtained (see Appendix B). The patient was then instructed on how to fill out the PHQ-9. While the patient

completed the PHQ-9, the researcher was available at the nurse's station to answer any questions. Upon completion, the PHQ-9 was collected by the researcher and taken to a locked security file cabinet.

### **Sample Size**

The sample size necessary for this study was determined by a power analysis conducted a priori under the guidance of the Office of Methodological and Data Sciences at Utah State University. The analysis was based upon an established alpha level = .05, an estimated power of 0.8 and a Cohens  $\delta$  of 0.5. The alpha was chosen due to the general acceptance among researchers and professionals that  $p = .05$  is a reliable indicator that a relationship between variables is statistically significant (Cohen, 2001). The  $\delta$  0.5 is considered moderate in evaluating relationships between variables and was thus chosen to be appropriate for this study. Using moderate power in determining the sample size also balances the risk of making Type I or Type II errors (Cohen, 2001). The sample size that was determined to be adequate for this study was 110.

### **Sample Characteristics**

Table 2 presents the demographic information for this study's participants. One hundred and eleven patients participated in the study-61 were male and 50 were female. The patients ranged in age from 22 to 95-years old. The diagnoses that were included in the study were chest pain, CHF, atrial fibrillation, pacemaker implants, MI, ACS, and rule out MI. The marital status categories used were married ( $n = 76$ ), single ( $n = 5$ ), widowed ( $n = 20$ ), and divorced ( $n = 8$ ).

Table 2

*Demographic Characteristics of the Sample*

Characteristics	<i>n</i>	Percent
Age (categories)		
0-44 years	5	4.5
45-60 years	22	19.8
61-70 years	14	12.6
71-80 years	37	33.3
81 or greater	33	29.7
Gender		
Male	61	54.9
Female	50	45.0
Diagnosis		
Chest pain	37	33.0
CHF	20	18.0
A. Fib	27	24.3
Pacemaker	9	8.1
MI	7	6.3
ACS	7	6.3
Rule out MI	4	3.6
Marital status		
Married	76	68.5
Single	5	5.4
Widowed	20	18.0
Divorced	8	8.1

### Instrumentation

The instrument selected to identify cardiac patients with depressive symptoms in this study is called the PHQ-9 (see Appendix A). This instrument was developed by Robert L. Spitzer, M.D., Janet B.W. Williams, M.D., and Kurt Kroenke, M.D., and is publically available online. The PHQ-9 is designed to facilitate the recognition and diagnosis of one of the most common mental disorders in primary care patients (i.e., depression). This instrument carries the added property to identify the severity of the depression for patients with a depressive disorder. A PHQ Depression Severity Index score can be calculated and repeated over time to monitor change. The brevity of this instrument makes it a useful clinical and research tool. The instrument consists of nine items aimed at assessing severity of depression by having participants indicate the frequency of their symptoms with a range of responses: not at all = 0, several days = 1, more than half the days = 2, and nearly every day = 3. A check is made by the appropriate level 0, 1, 2, or 3, for each question. The point value for each check is added up to give a total score. A score of 5-9 indicates mild depression, a score of 10-14 indicates moderate depression, a score of 15-19 indicates moderately severe depression, and a score of 20-27 indicates severe depression. Any patient scoring  $\geq 5$  was classified as depressed for the purposes of this study (Banazak, 2000).

The PHQ-9 has been validated and found to have concurrent validity with the BDI ( $r = .67, p < .001$ ). It also has been found to have had 1 month test-retest reliability ( $r = .894, p < .001$ ) and an internal consistency of items at 0.85 (Adewuya, Ola, & Afolabi, 2006). Using the receiver operating characteristic (ROC) curve, the optimal cut-off score for minor depressive disorder is 5 with a sensitivity of 0.897, specificity of 0.989, a positive predictive value of 0.875, a negative predictive value of 0.981, and overall correct classification rate of 0.973. MDD is 10 with a sensitivity of 0.846, a specificity of 0.994, a positive predictive value of 0.750, a negative

predictive value of 0.996, and OCC rate of 0.992 (Adewuya et al., 2006). Any patient scoring  $\geq 5$  was classified as depressed for the purposes of this study.

Kroenke, Spitzer, and Williams, (2001), conducted the PHQ Primary Care Study that was conducted from May 1997 to November 1998 to assess the reliability, efficiency, criterion validity, and construct validity of the PHQ-9. Three thousand patients presenting to five general internal medicine clinics and three family practice clinics participated in the study. Before seeing the physicians, the patients completed the PHQ-9. These same patients additionally completed the Medical Outcomes Study Short-Form General Health History (SF-20). The SF-20 measures functional status in six domains (all scores from 0 to 100; 100 = best health). Patients also estimated the number of physician visits and disability days during the past 3 months. Midway through the PHQ Primary Care Study, a Mental Health Professional (MHP) (a PhD clinical psychologist or one of three senior psychiatric social workers) attempted a phone interview with all entered subjects who agreed to participate within 48 hours of completing the PHQ-9. Agreement between the PHQ-9 and the MHP diagnosis was examined. The sensitivity was 73% and maintained a high specificity of 94%. The internal reliability of the PHQ-9 was calculated with a Cronbach's alpha of 0.89. The correlation between the PHQ-9 that the patient completed in the clinic and that administered over the telephone by the MHP within 48 hours was 0.84. The mean scores were nearly identical (5.08 vs. 5.03). As PHQ-9 scores increased, worsening functional ability ensued. Permission to use this instrument has been granted by Kurt Kroenke as this instrument is free for the public to use.

### **Pilot Testing**

The purpose of the pilot study was to discover limitations of the survey such as: understandability of the survey questions, time needed to complete the survey, and even limitations of the questions. The pilot study took place at an outpatient cardiac rehabilitation program located in a hospital in the intermountain west. Cardiac patients engaging in the outpatient cardiac rehabilitation program were asked to complete the PHQ-9 once informed consent had been obtained. Upon completion of the PHQ-9, the patients were asked to provide feedback regarding the questionnaire and its utility. A separate form (see Appendix C) was included with the PHQ-9 for each patient to complete to provide input about the questionnaire. This form had specific questions that were answered using a Likert scale. A comments section was also provided to obtain additional input not falling within these specific questions. The time necessary to complete the survey was determined from the pilot study and was approximately 10 minutes. The comments and input gained from the pilot study helped to direct the process in the actual study that was conducted on hospitalized cardiac patients.

Particular findings from the pilot study helped the researcher to modify the final study. The majority of patients participating in the pilot study felt that the PHQ-9 was easy to read and follow. It was discovered that patients required verbal reinforcement of the written instructions in order to be clear as to how to select the most appropriate answer for the items on the PHQ-9. In the actual study, this researcher did this with each participant.

### **Data Collection**

This researcher screened patients admitted as inpatients to one critical care unit in the intermountain west in accordance with Utah State University Institutional Review Board (IRB) guidelines and hospital policies that have already been approved by the hospital research review



board (see Appendix E). Evidence of cardiac patient status was determined through a daily review of a computerized list of all inpatients admitted to the facility having had a troponin blood test ordered. This test is given to any patient who is suspected of having an ACS. This researcher, administered the PHQ-9 to cardiac patients who have had CABG, mitral valve replacement (MVR), aortic valve replacement (AVR) surgery, a percutaneous transluminal coronary angioplasty (PTCA), stent placement, atrial fibrillation, CHF, unstable angina, ACS, MI, pacemaker implantation, and chest pain, and who have come to a stable point following their hospitalization.

This researcher determined if the patient met study inclusion criteria and if the patient's physician had agreed to allow the researcher to administer the PHQ-9. These physician permission forms were collected previously (see Appendix D). A list of these physicians was with the researcher when determining whether patients would be included in the study. The student researcher introduced herself to each patient and explained that she would like the patient to be a participant in a study on "Depression Detection in Hospitalized Cardiac Patients." Once the patient agreed to participate, informed consent was obtained. The patient was then instructed on how to complete the PHQ-9 and told that it should take approximately ten minutes to complete. Upon completion of the PHQ-9, the student researcher took the survey and delivered it to the designated research depository that had a locked file cabinet in the researcher's office.

This researcher then audited all charts of patients that scored  $\geq 5$  on the PHQ-9 following the patient's discharge from the hospital. This was done in order to be able to view all documentation in the completed chart. Patient charts were audited in order to assess the current practice habits for recognizing active depression in cardiac patients by their cardiac or internal medicine physician(s). Multiple methods were used to identify whether depressive symptoms were documented in the inpatient hospital chart. The methods included: (a) a diagnosis of depression in the patient chart, (b) a diagnosis of depression at the time of hospital discharge, (c)

evidence of active depression therapy (antidepressant medications started or dose adjusted of current medication, psychological counseling, or psychiatric consultation), and (d) evidence that depressive symptoms would be followed or management would be instigated as an outpatient.

Low-dose tricyclic antidepressants are frequently used for insomnia, neuropathic pain, and fibromyalgia, and Bupropion is often used for smoking cessation. The charts of patients taking these medications were reviewed to identify whether these medications were prescribed for an indication other than depression. Patients whose only criterion for recognition was the use of these medications and whose charts indicated that these medications were prescribed for a non-depressive disorder were classified as unrecognized.

### **Data Analysis**

The data from the questionnaires were entered into SPSS version 17.0 for analysis. Following data entry into the program, such data were randomly checked for accuracy regarding data entry. Statistical tests were run to answer the research questions (see Table 3).

The chi-square goodness of fit test was used to determine how well the obtained sample proportions fit the population proportions specified by the null hypothesis on research questions 2 and 4 (Gravetter & Wallnau, 2005). Logistic regression was used to answer research question 5 to

Table 3

*Research Questions and Data Analysis Procedures*

Research question	Measure	Data analysis
1. Are cardiologists and/or internal medicine physicians detecting depression in their cardiac patients during a hospitalization admission accurately as compared to the PHQ-9?	Patient records PHQ-9 $\geq 5$	Fisher's Exact Test
2. Are there differences in the rates of depression in the different diagnostic groups?	PHQ-9 $\geq 5$	Chi-Square Goodness of Fit Test
3. Are there differences between the rates of depression in male and female patients?	PHQ-9 $\geq 5$	Fisher's Exact Test
4. Are there differences between the rates of depression in patients who are married, single, widowed, or divorced?	PHQ-9 $\geq 5$	Chi-Square Goodness of Fit Test
5. Does the age of a patient predict the risk for depression following a cardiac event?	PHQ-9 $\geq 5$	Logistic Regression
6. Are there differences between the rates of depression in patients that have a prior cardiac diagnosis versus those with a new cardiac diagnosis?	PHQ-9 $\geq 5$	Fisher's Exact Test

determine whether the age of participants predicted depression among those patients. The Fisher's exact test was used to answer research question 1, 3, and 6 to determine whether proportions are significantly different from what is expected.

### Summary

This chapter discussed the methodology for this research study concerning depression detection in hospitalized cardiac patients. The topics that were covered were research design, sampling procedures, instrumentation, pilot study, data collection, and data analysis.

## CHAPTER IV

### RESULTS

This study was carried out to expand the research that has already been conducted by assessing the rate of detection of depression among hospitalized patients diagnosed with a wide spectrum of cardiac-related disorders. This chapter discusses the results of the six research questions introduced in Chapter I and III. The student researcher used SPSS 17.0 software in all of the analyses. The results are presented below.

#### **Question Number One**

Research question #1: *Are cardiologists and/or internal medicine physicians detecting depression in their cardiac patients during a hospital admission accurately as compared to the PHQ-9?* A total of 111 PHQ-9 surveys were administered to hospitalized patients with a suspected cardiac diagnosis. Eighty-three patients were identified as depressed and 28 patients were identified as nondepressed by the PHQ-9 (see Table 4). Of the 83 patients identified as depressed, only nine of those patients were identified as depressed by physicians as measured by their notes in the patients' charts. Physicians identified two patients as depressed that were not identified by the PHQ-9. A comparison of the patients identified as depressed by the PHQ-9 ( $83/111 = 75\%$  depression rate) and those identified by physicians ( $11/111 = 10\%$  depression rate) reveals different rates of detection. The fishers exact test revealed ( $p = .727$ , FET). The null hypothesis for the fishers exact test for independence states that the two variables being measured are not associated; that is, for each individual that the PHQ-9 identified depression, there is no association related to the physicians recognition of depression.

Table 4

*Patient Depression Detection*

	PHQ-9 identified depression				Total
	Yes	%	No	%	
Physician-identified depression	Yes	9 (11)	2 (7)		11
	No	74 (89)	26 (11)		100
	Total	83 (75)	28 (25)		111

This further supports the observed lack of physician identification of depression in their patients.

The sensitivity (i.e., true positive rate) for physician recognized depression is only 10.8% as noted by the PHQ-9.

### Question Number Two

Research question #2: *Are there differences between the rates of depression in the different diagnostic groups?* Each of the 111 patients fell into one of seven cardiac diagnostic groups (see Table 5). The highest rates of depression were identified in the MI group (7/7 = 100%) and the R/O MI group (4/4 = 100%). The next highest rates of depression were identified in the CHF group (19/20 = 95%) and the ACS group (5/7 = 71%). The lowest rates of depression were identified in the A Fib group (18/27 = 67%) and Pacer group (6/9 = 67%). Although rates of depression between diagnostic groups differed from one another, those differences were not statistically different from one another (chi-square = 11.271,  $p = .080$ ).

Table 5

*Depression Among Diagnostic Groups*

		PHQ-9 identified depression				Total
		Yes	%	No	%	
Cardiac diagnosis	CP	24	64.8	13	35.1	37
	CHF	19	95.0	1	5.0	20
	A. Fib	18	66.6	9	33.3	27
	Pacer	6	66.6	3	33.3	9
	MI	7	100.0	0	0	7
	ACS	5	71.4	2	28.5	7
	R/O MI	4	100.0	0	0	4
	Total	83	74.8	28	25.2	111

**Question Number Three**

Research question #3: *Are there differences between the rates of depression in male and female patients?* There were 61 males and 50 females that completed the PHQ-9. Of those 111 surveys, there were 44 males and 39 females that were identified as depressed (see Table 6). A comparison of the male patients identified as depressed ( $44/61 = 72\%$  depression rate) and female patients identified as depressed ( $39/50 = 78\%$  depression rate) revealed similar rates of detection ( $p = .517$ , FET).

Table 6

*Male and Female Depression*

Gender	Yes	%	No	%	Total
Male	44	(72.1)	17	(27.9)	61
Female	39	(78.0)	11	(22.0)	50

**Question Number Four**

Research question #4: *Are there differences between the rates of depression in patients who are married, single, widowed, or divorced?* The highest rates of depression were identified in the divorced patients ( $8/9 = 89\%$ ), the widowed patients ( $17/20 = 85\%$ ) and the single patients ( $5/6 = 83\%$ ) (see Table 7). The lowest rate of depression was found in the married patients

Table 7

*Marital Status and Depression*

Marital status	Yes	%	No	%	Total
Married	53	69.7	23	30.2	76
Single	5	83.3	1	16.7	6
Widowed	17	85.0	3	15.0	20
Divorced	8	88.9	1	11.1	9
Total	83	74.8	28	25.2	111

( $53/76 = 70\%$ ). A chi-square goodness of fit test indicated that the rates of depression between marital status groups did not differ significantly (chi-square = 6.060,  $p = .109$ ).

### Question Number Five

Research question #5: *Does the age of the patient predict the risk for depression following a cardiac event?* Logistic regression was used to answer this question. Age was used as a predictor variable and depression detection, as indicated on the PHQ-9, was used as the dependent variable in the model. The model accounted for 12% to 17% of the variance in depression detection. Table 8 gives coefficients, the Wald statistic, associated degrees of freedom, and *p*-values for age. The table indicates that age does not reliably predict depression among the patients in this sample ( $p = .251$ ).

Table 8

*Variables in the Equation*

	B	S.E.	Wald	df	Sig.	Exp(B)
Age	-0.017	0.015	1.316	1	0.251	0.983
Constant	0.137	1.077	0.016	1	0.899	1.146

### Question Number Six

Research question #6: *Are there differences between the rates of depression in patients that had a prior cardiac history versus those with a new diagnosis?* There were 66 patients with a prior cardiac diagnosis and 45 patients with a new cardiac diagnosis that completed the PHQ-9 (see Table 9). There were 53 patients with a prior cardiac diagnosis identified as depressed and 30 patients with a new cardiac diagnosis identified as depressed. A comparison of the patients with a prior cardiac diagnosis ( $53/66 = 80\%$  depression rate) and patients with a new cardiac diagnosis



(30/45= 67% depression rate) revealed rates of detection that were not statistically significantly different ( $p= .328$ , FET).

Table 9

*Prior and New Cardiac Diagnosis and Depression*

		PHQ-9 identified depression				
		Yes	%	No	%	Total
<hr/>						
Prior cardiac diagnosis						
Yes	53	80.3	13	19.7	66	
No	30	66.6	15	33.3	45	
Total	83	74.8	28	25.2	111	

### Summary

Chapter IV presented the results of the research questions posed in Chapters I and III. The next chapter will provide a discussion relating the results found in this study and a comparison to those found in past research. Suggestions for future research and the implications for the discipline of health education will also be explored.

## CHAPTER V

### DISCUSSION

#### **Introduction**

The current study was intended to determine whether cardiologists and/or internal medicine physicians are identifying depression in their patients admitted to the intensive care unit with a cardiac diagnosis. While past research has shown that physicians are not consistently identifying depression in their patients, these studies have been limited to very specific diagnostic groups (Amin et al., 2005; Feinstein et al., 2006; Hermann-Lingen, 2001; Simon et al., 2004; Thornton, 2002). Furthermore, a great deal of research exists that supports the notion that prolonged depression in the cardiac patient increases a patient's risk for further cardiac events (Barefoot & Schroll, 1996; Carney et al., 1996; Gehi et al., 2005; Parahk et al., 2008). This chapter will discuss the results of Chapter IV and the implications they may have for health educators. Suggestions will also be presented for future researchers.

#### **Research Question Number One**

Research question one was designed to determine whether those patients that scored  $\geq 5$  on the PHQ-9 were also being identified as depressed by the physician who was following their case during their hospital stay. This study found large differences between the rates of depression identified by the PHQ-9 (75%) and the rates of depression identified by physicians (10%). The fisher's exact test revealed no association between these variables. This further supports the idea that physicians are doing a poor job of identifying depression in their cardiac patients.

Prior research, however, has also indicated a gap. Thornton (2001) found that depression was missed in 50% of all cases. Of those 50%, less than 10% of these cases had any kind of treatment instituted. Feinstein et al. (2006) reported that in a national survey of 796

cardiovascular physicians, 84.8% believed that up to half of their patients with CHD had depression and yet 71% asked less than half of their patients with CHD about depression.

It could be argued that physicians are too busy to think about any medical problems other than those that they are there to directly diagnose and resolve. As managed care becomes more prevalent in the United States, so do the restrictions that physicians face regarding time spent with the patient. Documentation is becoming so cumbersome that many physicians may feel overwhelmed by the new rules and regulations that they must learn to implement from year to year. This process, unfortunately, is constantly changing as reimbursement issues become more complex. Cardiologists, it would seem, recognize the likely prevalence of depression among their patients, but don't see it as a part of their role to screen for depression, even though depression has a negative effect on cardiovascular outcomes. Cardiologists may deem this responsibility as belonging to the patient's primary care physician.

The disparity observed may just be a result of the difficulty in discriminating symptoms of depression from those related to other medical conditions, or co-morbidities (Davies et al., 2004). Somatic symptoms such as joint pain, limb pain, back pain, gastrointestinal pain, fatigue, psychomotor activity changes, appetite changes, diminished energy, and sleep disturbances, can be manifested by depression or by other medical illnesses (Goldstein, 2006; Trevioli, 2006). It may seem less difficult and less time consuming to simply treat the somatic symptoms that a patient is experiencing. Determining why a patient is depressed can be a complicated and time-consuming process. To get to the core of some problems may be far beyond the scope of care with which a physician is comfortable. A physician may be afraid of the liability that he/she might face if a diagnosis of depression is given. If a physician identifies depression, then wouldn't it also be his/her responsibility to make sure that the patient be treated for this problem? From an ethical standpoint, a physician should feel it his responsibility to treat depression if discovered. It would, therefore, be advantageous for a screening tool to be administered prior to a

patient meeting with the physician so that a depression score could then be communicated with the physician and the issue addressed. The physician could then refer the patient to another provider in order to ensure that the patient received the help that he/she needs. Unfortunately, somatic symptoms may be the only way that some populations express the presence of an emotional problem, especially if depression carries a cultural or group stigma (Rosenthal, 2003).

This study was conducted in a smaller U.S. city and therefore the hospital is considered to be a community hospital. Physicians in a smaller community hospital setting may be more inclined to follow certain rules set by the powers that be. Depending on what those rules are, the possibility of “stepping on the other guy’s toes” or “overstepping boundaries” is very real and may be a social moray that must be followed in order to be accepted or respected.

Therefore, it remains that physician detection of depression in cardiac patients is under represented. The factors that appear to be causing this under representation are physician time restraints, documentation issues, guidelines and regulations, and possibly liability issues. A physician’s tendency to treat a patient’s somatic symptoms first may also represent either their lack of experience or the hesitation to delve in to the realm of treating mental health issues.

## **Research Question Number Two**

Research question number two asked: are there differences between the rates of depression in the different diagnostic groups? The current study has MI and CHF included in its diagnostic groups and has added CP, A fib, Pacer, ACS, and R/O MI. Of all of these diagnostic groups combined, it was found that the rates of depression range anywhere from 67-100%. No statistically significant differences between groups were found. Notably, the rates of depression identified in this study were higher than previous research which showed depression ranging from 15-65% (Amin et al., 2005; Feinstein et al., 2006; Hermann-Lingen, 2001; Kaptein et al., 2005), possibly because the PHQ-9 captures mild, moderate and severe depression when utilizing the  $\geq 5$

cutoff score. Additionally, past research made use of different instruments such as the BDI (Lesperance, & Frasure-Smith et al., 2000; Kaptein et al., 2005), Hospital Anxiety and Depression Scale (HADS) (Murphy et al., 2008), and the CES-D (Ariyo et al., 2000), among others, along with different cutoff scores, making comparisons to the results of this study somewhat problematic. Finally, most studies included patients from only one diagnostic group making comparisons of the rates of depression between patients with differing diagnoses also very problematic.

Therefore, it appears difficult to gain any meaningful comparisons from past research with this study. Although this study seemed to show that amongst diagnostic groups, depression remains high if you start measuring beginning with mild depression and ending with severe depression

### **Research Question Number Three**

Research question three was designed to detect whether there were differences in the rates of depression between men and women who had been hospitalized with a cardiac diagnosis. This study found that about 72% of males were identified as depressed and 78% of females were identified as depressed. Although more women were identified as being depressed at a higher rate than were men, the difference was not statistically significant in this study. Faller et al. (2007) found no gender difference in rates of depression detected among heart failure patients. Alternatively, Naqvi et al. (2007) found that women did suffer higher rates of depression than did men after experiencing ACS.

Danielsson and Johansen (2005) have suggested that men and women convey their experiences of depression differently, which may result in artificially higher rates of depression detected among women than among men. This phenomenon is made manifest with men speaking more easily about physical distress than emotions, whereas women speak more readily about

emotional distress, shame and guilt. Upon examination of the PHQ-9 it appears that of the nine questions asked, six questions have more of a physical component to them and only three questions have more of an emotional component to them.

Therefore, it might appear that men would score higher as a whole on this questionnaire than would women. This, however, did not hold true for this study. Using surveys that are more specific to gender might yield better results.

#### **Research Question Number Four**

Research question four was designed to determine whether there were differences in the rates of depression in patients who are married, single, widowed, or divorced. In this study, no statistically significant differences were observed.

Prior research has come to the same conclusion. Chung et al. (2009) also found that levels of depressive symptoms were similar between married and nonmarried heart failure patients (10.9 vs. 12.1%). Panagiotakos et al. (2008) found that hospitalized patients who were married or nonmarried had very similar rates of depression (20.4% and 22.8%).

Research seems to point in the same direction as the results of this study. It may be that some people who are married have better social support than other married people and the same would be true in the nonmarried groups. Similarly, social support may decrease depression in all of the marital status groups. Social support, personality characteristics, and close relationships may have more to do with depression than marital status. Marital status cannot be used as a proxy for social support. Furthermore, we do not know what kind of depression a patient actually has. It could be inherited, it could be chronic, or it could be acute.

#### **Research Question Number Five**

Question five was designed to determine whether the age of the patient predicts the risk for depression. This study found that age did not reliably predict depression in cardiac patients.

Prior research seems to find the same kind of results. The basic difference occurs when aging is associated with different types of disability, comorbidities and functional impairment. Healthy, normally functioning older adults are at no greater risk for depression than younger adults (Jorm, 2000; Roberts, Kaplan, Shema & Strawbridge, 1997). This seems to be linked to the idea that an increase in comorbidities would also be linked to increased depression regardless of age.

Personality traits may play a big role in whether a person becomes depressed. An optimistic older individual may fare much better than a pessimistic younger adult, regardless of comorbidities.

Bremmer et al. (2006) made a connection between older age predicting first cardiac events. Depressive disorders, and in particular, MDD in late life (75 years and greater) were mediated by subclinical atherosclerosis, which would confine the diagnoses to ischemic heart diseases.

Increased age, indeed, may have very little to do with depression. The things that are more likely to predict depression could be things like marital status, education level, financial strain, chronic medical conditions, functional impairment, cognitive problems, life events, neighborhood problems, social isolation, and social support.

### **Research Question Number Six**

Question six was designed to detect whether there were differences between the rates of depression in patients that had a prior cardiac history versus those with a new diagnosis. In this study, no difference was observed at a statistically significant level. This researcher was unable to find any prior research that examined depression among these two groups.

It may seem that a patient with a prior cardiac history has more reasons to be depressed, especially if long-term disability has been an issue. On the other hand, if a patient has been

through a cardiac event before, he/she may feel more comfortable with the situation and have come to terms with the presence of heart disease in general. They may feel that they have an “edge” on knowing how to recognize symptoms of a problem earlier, thus helping them to be able to take care of things in a more timely matter. They may have taken the time to educate themselves regarding their cardiac problems and made lifestyle modifications that make them feel more empowered and able to manage their disease effectively.

It is very possible that personality characteristics could have something to do with how a patient may respond to a new diagnosis versus a prior history of cardiac problems. A person that possesses the characteristics of optimism, may fair better than an individual that possessed the characteristic of pessimism in whatever group they might be a part of. A hopeful patient may fair better than a hopeless patient.

For those individuals who come in with what would be classified as a new diagnosis, there are many arguments for and against depression. A person who has never had any diagnosis of heart disease may not have any idea what is really happening to him/her and are not suspect of his/her heart in general. If the diagnosis shows that the problem really does lie in their heart, they may find the situation depressing from the standpoint of not knowing what to expect in the future. Lack of understanding and education can be one of the biggest hurdles to conquer. As these individuals gain more knowledge about their condition, the fear and depression will generally subside. If the individual does not have the desire, time, or ability to gain more education about his/her condition, they may continue to have or it may result in higher levels of depression.

Ultimately, it would seem that prior versus new cardiac diagnosis has very little to do with depression. Personality characteristics, past experience, environmental conditioning, socioeconomic level, comorbidities, and social support among others, would seem to have more influence on depression than prior versus new cardiac diagnosis.



### **Limitations of the Study**

The results of this study revealed information that may be helpful to health education professionals in the future. The study was designed to detect whether physicians are detecting depression in their patients that score  $\geq 5$  on the PHQ-9 questionnaire. It was shown in this study that out of 83 patients that scored high enough to be categorized as depressed on the PHQ-9, only nine of those patients were identified by physicians.

Depression inventories are easy to administer and there are many in publication that are free to use and have good validity and reliability. These inventories can be a simple, yet effective way of identifying cardiac patient's who are depressed. Many of these inventories are currently being used in many health care settings. They are not only used, but are required by some governing bodies such as The American Association of Cardiovascular and Pulmonary Rehab (AACVPR) on a national level within all cardiac and pulmonary rehab programs.

In the hospital setting, a depression inventory could easily be given by a myriad of qualified practitioners, including health educators. The physician need not be bothered except to be informed of the patients score and possibly the severity of depression based on results from the inventory. The physician may then choose to address the situation while the patient is in the hospital or to simply follow up when the patient comes to the office for his/her follow-up appointment in approximately 2 to 4 weeks post discharge. At the appointment, the inventory could be given to the patient to fill out with any required initial paperwork. If depression persists, the physician could then begin to address the problem.

It appears that more emphasis needs to be given to depression detection in the cardiac patient from the moment of hospitalization and beyond. This is due to the fact that the longer depression persists, the higher the risk becomes for future cardiac events. In the process of treating the whole patient, including any depressive symptoms, many of the somatic symptoms

might also be alleviated.

In our current society, health care reform and cost containment have become increasingly more important issues that are currently being addressed by the United States government. It would seem that treating underlying psychosocial issues, such as depression, would not only be directly beneficial to the patient, but also would result in lower costs in the long term.

### **Implications for Health Education**

There were limitations to the present study that could be corrected by future researchers. The present study had a relatively small sample size that was not random in nature. The sample was a convenience sample. With a larger sample, more patients with the same diagnosis would be viewed and the percentages may be more even and not as high in some groups.

Unfortunately, the physician sample size was also very small. There were only two cardiologists who practice at the facility where the study was being conducted. Therefore, there was not a great deal of differing attitudes. Furthermore, both types of physicians, cardiologists and internal medicine, very infrequently even discuss depression in their notes. Although there were quite a few more internal medicine physicians, the sample was still smaller than would be ideal.

Self-report surveys carry with them many limitations. The major limitation of the survey method is that it relies on a self-report method of data collection. Intentional deception, poor memory, or misunderstanding of the question can all contribute to inaccuracies in the data. Furthermore, this method is descriptive, not explanatory, and, therefore, cannot always offer any insights into cause-and-effect relationships. This may be corrected by the patient being interviewed by a professional in the field of psychology, and yet you would still have the problem of whether or not the patient would feel comfortable talking to a professional. Beyond this, the

patient may have assumptions already formed that would cause them to not give truthful answers to the questions.

A restriction of range is a definite possibility in this study, thereby limiting or reducing the size of the relationship between some factors and depression. The sample was skewed when looking at age, marital status, and diagnostic group. This may have reduced the size of the relationships found in this study. Future research could stratify the sample by group status so that the numbers of participants in each group would be similar. This might make the percentages of depression in each diagnostic group more representative.

It is clear from prior work that depressed cardiac patients have poorer health-outcomes than do non-depressed patients. Studies in the future could also attempt to break down the cost differences, if any, between the care provided for a depressed versus non-depressed cardiac patient.

### **Summary**

This chapter has discussed the results of the analyses conducted on the data. Implications for health educators, as well as recommendations for future research have been proposed. This study found large differences between the rates of depression identified by the PHQ-9 (75%) and the rates of depression identified by physicians (10%).

With the addition of more diagnostic groups, the study was expanded. In general, rates of depression in this study were higher in all groups than that found in prior research. Despite no statistical difference being achieved, this study suggests that depression rates may be higher than recently discovered. Although more women were identified as being depressed at a higher rate than men, the difference was not statistically significant in this study. Age did not reliably predict depression in cardiac patients.

## REFERENCES

- Adewuya, A., Ola, B., & Afolabai, O. (2006). Validity of the patient health questionnaire (PHQ9) as a screening tool for depression amongst Nigerian university students. *Journal of Affective Disorders*, 96, 89-93.
- Ali, S., Gehi, A., Na, B., & Whooley (2007). Self reported medication adherence and cardiovascular events in patients with stable coronary heart disease: The heart and soul study. *Archives of Internal Medicine*, 167, 1798-1803.
- Almeida, O., & Pfaff, J. (2005). Depresssion and smoking amongst older general practice patients. *Journal of Affective Disorders*, 86, 317-321.
- Amin, A., Jones, M., Nugent, R., Rumsfeld, J., & Spertus, A. (2005). The prevalence of unrecognized depression in patients with acute coronary syndrome. *American Heart Journal*, 152, 928-934.
- Amital, A., (2008, February 8). Serious life events among resistant and non resistant MDD patients. *Journal of Affective Disorders*. Retrieved February 3, 2008, from <http://www.ncbi.nlm.nih.gov/sites/entrez>.
- Anisman, H., Merali, Z., & Stead, J. (2008, March 18). Experimental and genetic contributions to depressive and anxiety like disorders: Clinical and experimental studies. *Neuroscience* [Epub ahead of print].
- Ariyo, A., Haan, M., Tangen, C., Rutledge, J., Cushman, M., Dobs, A., et al. (2000). Depressive symptoms and risks of coronary heart disease and mortality in elderly Americans. *Circulation*, 102, 1773.
- Asnis, G., Sachar, E., Halbresich, U., Nathan, R., Novacenko, H., & Ostrow, L. (1981). Cortisol secretion in relation to age in major depression. *Psychosomatic Medicine*, 43, 235-242.
- Banazak, D. (2000). Minor depression in primary care. *Journal of the American Osteopath Association*, 100, 783-787.
- Barefoot, J., & Schroll, M. (1996). Symptoms of depression, acute myocardial infarction, and total mortality in a community sample. *Circulation*, 93, 1976-1980.
- Bhagwagar, Z., Hafizi, S., & Cowen, P. (2003). Increase in concentration of waking salivary cortisol in recovered patients with depression. *American Journal of Psychiatry*, 160, 1890-1891.
- Blumenthal, J., Lett, H., Babyak, A., White, W., Smith, P., & Mark, D. (2003). Depression as a risk factor for mortality after coronary artery bypass surgery. *Lancet*, 362, 604-609.
- Boden-Albala, B., Litwak, E., Elkind, M., Rundek, T., & Sacco, R. (2005). Social isolation and outcomes post stroke. *Neurology*, 63, 1888-1892.

- Bremmer, M., Hoogendijk, W., Deeg, D., Shoeners, R., Schalk, B., & Beekman, A. (2006). Depression in older age is a risk factor for first ischemic cardiac events. *American Journal of Geriatric Psychiatry*, 14, 523-530.
- Burker, E., Blumental, J., Feldman, M., Burnett, R., White, W., & Smith, L. (1995). Depression in male and female patients undergoing cardiac surgery. *British Journal of Psychology*, 34, 119-128.
- Carney, R., Freedland, K., Sheline, Y., & Weiss, E. (1996). Depression and coronary heart disease: A review for cardiologists. *Clinical Cardiology*, 20, 196-200.
- Cervilla, J., Molina, E., Rivera, M., Torres-Gonzales, F., Bellon, J., & Moreno, B. (2007). The risk for depression conferred by stressful life events is modified by variation at the serotonin transporter 5HTTLPR genotype: Evidence from the Spanish PREDICT-gene cohort. *Molecular Psychiatry*, 12, 748-755.
- Chen, Y., Chen, M., Chou, F., Sun F., Chen, P., & Tsai, K. (2007). The relationship between quality of life and posttraumatic stress disorder or major depression for firefighters in Kaohsiung, Taiwan. *Quality of Life Research*, 16, 1289-1297.
- Choi, N., & McDougall, G. (2007). Comparison of depressive symptoms between homebound older adults and ambulatory older adults. *Aging Mental Health*, 11, 310-322.
- Chung, M., Wu, J., Dekker, R., Lennie, T., Moser, D., & Riegel, B (2009). Marital status as an independent predictor of event free survival of patients with heart failure. *American Journal of Critical Care*, 18, 562-570.
- Cohen, B. (2001). *Explaining psychological statistic* (2<sup>nd</sup> ed.). New York: Wiley.
- Connerney, I., Shapiro, P., McLaughlin, J., Bagiella, E., & Sloan, R. (2001). Relation between depression after coronary artery bypass surgery and 12-month outcome: A prospective study. *Lancet*, 11, 1766-1771.
- Coruuble, E. (2006). Recurrent depression and life events: ACTUEL survey. *Encephale*, 32, 983-987.
- Danielsson, U., & Johansson, E. (2005). Beyond weeping and crying: A gender analysis of expressions of depression. *Scandinavian Journal of Primary Health Care*, 23, 171-177.
- Danner, M., Kasi, S., Abramson, J., & Vaccarino, V. (2003). Association between depression and elevated c-reactive protein. *Psychosomatic Medicine*, 65, 347-356.
- Davies, S., Jackson, P., Potokar, J., & Nutt, D. (2004). Treatment of anxiety and depressive disorders in patients with cardiovascular disease. *British Medical Journal*, 13, 364-367.
- Davis, F. (1978). *Tabers cyclopedic medical dictionary*. Philadelphia: F.A. Davis.

- De Jonge, P., Denollet, J., Van Melle, J., Kuyper, A., Honig, A., Schene, A., et al. (2007). Associations of type D personality and depression with somatic health in myocardial infarction patients. *Journal of Psychosomatic Reserves*, 63, 477-482.
- Denollet, J., Pedersen, S., Vrints, C., & Conraads, V. (2006). Usefulness of type D personality in predicting five year cardiac events above and beyond concurrent symptoms of stress in patients with coronary heart disease. *American Journal of Cardiology*, 97, 970-973.
- Denollet, J., Sys, S., Stroobant, N., Rombouts, H., Gillebert, T., & Brutsaert, D. (1996). Personality as independent predictor of long term mortality in patients with coronary heart disease. *Lancet*, 347, 417-421.
- Denollet, J., Vaes, J., & Brutsaert, T. (2000). Inadequate response to treatment in coronary heart disease: Adverse effects of type D personality and younger age on 5-year prognosis and quality of life. *Circulation*, 102, 630-635.
- Dickens, C., Percival, C., McGowan, L., Douglas, J., Tomenson, B., & Cotter, L. (2004). The risk factors for depression in first myocardial infarction patients. *Psychological Medicine*, 34, 1083-1092.
- Diener, C., Kuehner, C., Brusniak, W., Struve, M., & Flor, H. (2008). Effects of stressor uncontrollability on psychophysiological, cognitive and behavioral responses in patients with major depression and dysthymia. *Psychological Medicine*, 8, 1-10.
- Douglas, K., Taylor, A., & O'Malley, P. (2004). Relationship between depression and c-reactive protein in a screening population. *Psychosomatic Medicine*, 66, 679-683.
- Dressler, W., Balieiro, M., Ribiero, R., & Dos Santos, J. (2006). Depressive symptoms and c-reactive protein in a Brazilian urban community. *Brazilian Journal of Medical and Biological Research*, 39, 1013-1019.
- Ehrman, J., Gordon, P., Visich, P., & Keteyian, S. (2003). *Clinical exercise physiology*. Champagne, IL: Human Kinetics.
- Elovaino, M., Jokela, M., Kivimaki, M., Pulkki-Raback, L., Lehtimaki, T., & Airla, N. (2007). Genetic variants in the DRD2 gene moderate the relationship between stressful life events and depressive symptoms in adults: Cardiovascular risk in young Finns study. *Psychosomatic Medicine*, 69, 391-395.
- Faller, H., Stork, S., Schowalter, M., Steinbuchel, T., Wollner, V., & Ertl, G. (2007). Depression and survival in chronic heart failure: Does gender play a role? *European Journal of Heart Failure*, 9, 1018-1023.
- Feinstein, R., Blumenfield, M., Orlowski, B., Frishman, W., & Ovanessian, S. (2006). A national survey of cardiovascular physicians' beliefs and clinical care practices when diagnosing and treating depression in patients with cardiovascular disease. *Cardiology in Review*, 14, 164-169.
- Ford, D., & Erlinger, T. (2004). Depression and c-reactive protein in US adults. *Archives of*

*Internal Medicine*, 164, 1010-1014.

- Frasure-Smith, N. (2000). Depression in patients with cardiac disease: A practical review. *Journal of Psychosomatic Research*, 48, 379-391.
- Freedland, K., Rich, M., Skala, J., Carney, R., Davila-Roman, V., & Jaffe, A. (2003). Prevalence of depression in hospitalized patients with heart failure. *Psychosomatic Medicine*, 65, 119-128.
- Gehi, A., Ali, S., Na, B., & Whooley, M. (2007). Self reported medication adherence and cardiovascular events in patients with stable coronary heart disease: The Heart and Soul Study. *Archives of Internal Medicine*, 167, 1798-1803.
- Gehi, A., Haas, D., Pipkin, S., & Whooley, M. (2005). Depression and medication adherence in outpatients with coronary heart disease: Findings from the Heart and Soul Study. *Archives of Internal Medicine*, 165, 2504-2513.
- Glassman, A. (2007). Depression and cardiovascular comorbidity. *Dialogues in Clinical Neuroscience*, 9, 9-17.
- Goldstein, M. (2006). Depression: An independent risk factor for cardiovascular disease. *Journal of the American Academy of Physicians*, 19, 40-46.
- Gott, M., Barnes, S., Parker, C., Payne, S., Seamark, D., Gariballa, S., et al. (2006). Predictors of the quality of life of older people with heart failure recruited from primary care. *Age and Aging*, 35, 172-177.
- Gravetter, F., & Wallnau, L. (2005). *Essentials of statistics for the behavioral sciences* (5<sup>th</sup> ed.). Belmont, CA. Thomsen Learning.
- Greaves, C. (2006). Effects of creative and social activity on the health and well-being of socially isolated older people: Outcomes from a multi-method observational study. *Journal of the Royal Society of Health*, 126, 134-142.
- Grigoriadis, S., & Robinson, G. (2007). Gender issues in depression. *Annals of Clinical Psychiatry*, 19, 247-255.
- Heo, S., Moser, D., & Widener, J. (2007). Gender differences in the effects of physical and emotional symptoms on health related quality of life in patients with heart failure. *European Journal of Cardiovascular Nursing*, 6, 146-152.
- Herrmann-Lingen, C. (2001). Anxiety and depression in cardiology patients: How to diagnose, how to treat? *Herz*, 26, 326-34.
- Huffman, J., Smith, F., Blais, M., Beiser, M., Januzzi, J., & Fricchione, G. (2006). Recognition and treatment of depression and anxiety in patients with acute myocardial infarction. *American College of Cardiology*, 98, 661-663.

- Husky, M., Mazure, C., Paliwal, P., & McKee, L. (2008). Gender differences in the co morbidity of smoking behavior and major depression. *Drug and Alcohol Dependency*, 93(1-2), 176-179.
- Intermountain Health Care. (2005). Living with heart failure. *Clinical Education Services*.
- Januzzi, J., & Pasternak, R. (2002). Depression, hostility, and social isolation in patients with coronary heart disease. *Current Treatment Options in Cardiovascular Medicine*, 4(1), 77-85.
- Jiang, W., Alexander, J., Christopher, E., Kuchibhalta, M., Gaulden, L., & Cuffe, M. (2001). Relationship of depression to increased risk of mortality and re-hospitalization in patients with congestive heart failure. *Archives of Internal Medicine*, 161, 1849-1856.
- Jorm, A. (2000). Does old age reduce the risk of anxiety and depression? A review of epidemiological studies across the adult life span. *Psychological Medicine*, 30(1), 11-22.
- Kaptein, K., De Jonge, P., Van Den Brink, R., & Korf, J. (2006). Course of depressive symptoms after myocardial infarction and cardiac prognosis: A latent class analysis. *Psychosomatic Medicine*, 8, 662-668.
- Karlsson, M., Edstrom-Pluss, C., Held, C., Henriksson, P., Billing, E., & Wallen, N. (2007). Effects of expanded cardiac rehabilitation on psychosocial status in coronary artery disease with focus on type D characteristics. *Journal of Behavioral Medicine*, 30, 253-261.
- Kemp, D., Malhorta, S., Franco, K., Tesar, G., & Bronson, D. (2003). Heart disease and depression: Don't ignore the relationship. *Cleveland Clinic Journal of Medicine*, 70, 745-761.
- Kendler, K., Karkowski, L., & Prescott, C. (1999). Causal relationship between stressful life events and the onset of major depression. *American Journal of Psychiatry*, 156, 837-841.
- Klein, D., Lewinsohn, P., Seeley, J., & Rohde, P. (2001). A family study of major depressive disorder in a community sample of adolescents. *Archives of General Psychiatry*, 58, 13-20.
- Kroenke, K., Spitzer, R., & Williams, J. (2001). The PHQ-9: Validity of a brief depression severity measure. *Journal of General Internal Medicine*, 16, 606-613.
- Krzyzkowiak, W. (2007). Depression after myocardial infarction and its psychosocial-conditions. *Archives of Polish Psychiatry*, 41, 679-691.
- Ladwig, K., Marten-Mittag, B., Lowel, H., Doring, A., & Koenig, W. (2005). C-reactive protein, depressed mood, and the prediction of coronary heart disease in initially healthy men: Results from the MONICA-KORA Augsburg Cohort Study. *European Heart Journal*, 26, 2537-2542.
- Lane, D., Carroll, D., & Lip, G. (1999). Psychology in coronary care. *Quarterly Journal of*



*Medicine*, 92, 425-431.

- Lauzon, C., Beck, C., Huynh, T., Dion, D., Racine, N., & Carignan, S. (2003). Depression and prognosis following hospital admission because of acute myocardial infarction. *Canadian Medical Association Journal*, 168, 547-552.
- Lederbogen, F., Gilles, M., Maras, A., Hamann, B., Colla, M., Heuser, I., et al. (2001). Increased platelet aggregability in major depression? *Psychiatry Research*, 102, 255-261.
- Lehto, S., Koukkinen, H., Hintikka, J., Viinamaki, H., Laakso, M., & Pyorala, K. (2000). Depression after coronary heart disease events. *Scandinavian Cardiovascular Journal*, 34, 580-583.
- Lesperance, F., & Frasere-Smith, N. (2000). Depression in patients with cardiac disease: A practical review. *Journal of Psychosomatic Research*, 48, 379-391.
- Lyons, M., Hitsman, B., Xian, H., Panizzon, M., Jerskey, B., & Santangelo, S. (2008). *Nicotine and Tobacco Research*, 10, 97-108.
- Magruder-Habib, K., Zung, W., & Fuessner, J. (1990). Improving physicians' recognition and treatment of depression in general medical care. *Medical Care*, 28, 239-250.
- Marinic, T., Ozvacic, Z., Katic, M., Polasek, O., & Lazic, D. (2007). Characteristics of depression in Otocac ten years after the war. *Acta Med Croatica*, 61, 25-31.
- Mitchell, R., Robertson, E., Harvey, P., Nolan, R., Rodin, G., Romans, S., et al. (2005). Sex differences in depression after coronary artery bypass graft surgery. *The American Heart Journal*, 150, 1017-1025.
- Moller-Leimkuhler, A. (2007). Women with coronary artery disease and depression: A neglected risk group. *World Journal of Biological Psychiatry*, 18, 1-10.
- Mondimore, F., Zandi, P., Mackinnon, D., McInnis, M., Miller, E., Crowe, R., et al. (2006). Familial aggregation of illness chronicity in recurrent, early-onset major depression pedigrees. *American Journal of Psychiatry*, 163, 1554-1560.
- Moser, D., Doering, L., & Chung, M. (2005). Vulnerabilities of patients recovering from an exacerbation of chronic heart failure. *American Heart Journal*, 150, 984.
- Munafo, M., Hitsman, B., Rende, R., Metcalfe, C., & Niaura, R. (2008). Effects of progression to cigarette smoking on depressed mood in adolescents: Evidence from the National Longitudinal Study of Adolescent Health. *Addiction*, 103, 162-171.
- Murphy, B., Elliott, P., Worcester, M., Higgins, R., Le Grande, M., Roberts, S., et al. (2008). Trajectories and predictors of anxiety and depression in women during the 12 months following an acute cardiac event. *British Journal of Health Psychology*, 13, 135-153.

- Musselman, D., Tomer, A., Manatunga, A., Knoght, B., Porter, M., Kasey, L., et al. (1996). Exaggerated platelet reactivity in major depression. *American Journal of Psychiatry*, 153, 1313-1317.
- Naqvi, T., Rafique, A., Andreas, V., Rahban, M., Mirocha, J., & Naqvi, S. (2007). Predictors of depressive symptoms post acute coronary syndrome. *Gender and Medicine*, 4, 339-351.
- Nierenberg, A., Trivedi, M., Fava, M., Biggs, M., Shores-Wilson, K., Wisniewski, S., et al. (2007). Family history of mood disorder and characteristics of major depressive disorder: A STAR\*D (sequenced treatment alternatives to relieve depression) study. *Journal of Psychiatric Research*, 41, 214-221.
- Otte, C., Marmar, C., Pipkin, S., Moos, R., Browner, W., & Whooley, M. (2004). Depression and 24 hour urinary cortisol in medical outpatients with coronary heart disease: The Heart and Soul Study. *Biological Psychiatry*, 56, 241-247.
- Pangiotakos, D., Pitsavos, C., Kogias, Y., Mantas, Y., Zombolos, S., Antonoulas, A., et al. (2008). Marital status, depressive episodes, and short term prognosis of patients with acute coronary syndrome: Greek study of acute coronary syndrome (GREECS). *Neuropsychiatric Disorders Treatment*, 4, 425-432.
- Parakh, K., Thombs, B., Fauerbach, J., Bush, D., & Ziegelstein, R. (2008). Effect of depression on late (8 years) mortality after myocardial infarction. *American Journal of Cardiology*, 101, 602-606.
- Pedersen, S., & Denollet, J. (2004). Validity of the type D personality construct in Danish post MI patients and healthy controls. *Journal of Psychosomatic Research*, 57, 265-272.
- Pedersen, S., Denollet, J., Van Gestel, Y., Serruys, P., & Van Domburg, R. (2008). Clustering of psychological risk factors enhances the risk of depressive symptoms 12 months post percutaneous coronary intervention. *European Journal of Cardiovascular Preventive Rehabilitation*, 15, 203-209.
- Pihl, E., Jacobsson, A., Fridlund, B., Stromberg, A., & Martensson, J. (2005). Depression and health related quality of life in elderly patients suffering from heart failure and their spouses: A comparative study. *European Journal of Heart Failure*, 7, 583-589.
- Pruessner, M., Hellhammer, D., Pruessner, J., & Lupien, S. (2003). Self reported depressive symptoms and stress levels in healthy young men: Associations with the cortisol response to awakening. *Psychosomatic Medicine*, 65, 92-99.
- Puig-Antich, J., Goetz, D., Davies, M., Kaplan, T., Davies, S., Ostrow, L., et al. (1989). A controlled family history study of prepubertal major depressive disorder. *Archives of General Psychiatry*, 46, 406-418.
- Rafanelli, C., Roncuzzi, R., & Milaneschi, Y. (2006). Minor depression as a cardiac risk factor after coronary artery bypass surgery. *Psychosomatics*, 47, 289-295.

- Roberts, R., Kaplan, G., Shema, S., & Strawbridge, W. (1997). Does growing old increase the risk for depression? *American Journal of Psychiatry*, 154, 1384-1390.
- Romanelli, J., Fauerbach, J., Bush, D., & Ziegelstein, R. (2002). The significance of depression in older patients after myocardial infarction. *Journal of the American Geriatrics Society*, 50, 817-822.
- Romans, S., Tyas, M., Cohen, M., & Silverstone, T. (2007). Gender differences in the symptoms of major depressive disorder. *Journal of Nervous Mental Disorders*, 195, 905-911.
- Rosenthal, M. (2003). Barriers to diagnosis, advances in therapy. *Journal of Open Access*, 4, S2-S9.
- Ruo, B., Rumsfeld, J., Hlatky, M., Liu, H., Browner, W., & Whooley, M. (2003). Depressive symptoms and health related quality of life: The Heart and Soul Study. *Journal of the American Medical Association*, 290, 215-221.
- Ryan, J., Carriere, I., Ritchie, K., Stewart, R., Toulemonde, G., Dartigues, J., et al. (2003). Late-life depression and mortality: Influence of gender and antidepressant use. *British Journal of Psychiatry*, 192, 12-18.
- Scherer, M., Himmel, W., Stanske, B., Scherer, F., Koschack, J., Kochen, M., et al. (2007). Psychological distress in primary care patients with heart failure: A longitudinal study. *British Journal of General Practice*, 57, 801-807.
- Schiffer, A., Pedersen, S., Widdershoven, J., Hendriks, E., Winter, J., & Denollet, J. (2005). The distressed (type D) personality is independently associated with impaired health status and increased depressive symptoms in chronic heart failure. *European Journal of Cardiovascular Preventive Rehabilitation*, 12, 341-346.
- Serebruany, V., Glassman, A., Malinin, A., Sane, D., Finkel, M., Krishnan, R., et al. (2003). Enhanced platelet/endothelial activation in depressed patients with acute coronary syndromes: Evidence from recent clinical trial. *Blood Coagulation and Fibrinolysis*, 14, 563-567.
- Steptoe, A., Owen, N., Kunz-Ebracht, S., & Brydon, L. (2004). Loneliness and neuroendocrine, cardiovascular, and inflammatory stress responses in middle-aged men and women. *Psychoneuroendocrinology*, 29, 593-611.
- Suarez, E. (2004). C-reactive protein is associated with psychological risk factors of cardiovascular disease in apparently healthy adults. *Psychosomatic Medicine*, 66, 684-691.
- The Criteria Committee of the New York Heart Association. (1994). *Nomenclature and criteria for diagnosis of diseases of the heart and great vessels* (9<sup>th</sup> ed). Boston: Brown.
- Thornton, L. (2001). Depression in post acute myocardial infarction patients. *Journal of the American Academy of Nurse Practitioners*, 13, 364-367.

- Tozzi, F., Prokopenko, I., Perry, J., Kennedy, J., McCarthy, A., Holsboer, F., et al. (2008). Family history of depression associated with younger age of onset in patients with recurrent depression. *Psychological Medicine*, 2, 1-9.
- Trivedi, M. (2004). The link between depression and physical symptoms. *Primary Care Companion of the Journal of Clinical Psychiatry*, 6, 12-16.
- Vaccarino, V., Lin, Z., Kasi, S., Mattera, J., Roumanis, S., Abramson, J., et al. (2003). Gender differences in recovery after coronary artery bypass surgery. *Journal of the American College of Cardiology*, 41, 307-314.
- Van den Broek, K., Martens, E., Nyklicek, I., Van der Voort, P., & Pedersen, S. (2007). Increased emotional distress in type-D cardiac patients without a partner. *Journal of Psychosomatic Reserves*, 63, 41-49.
- Vanderhorst, R., & McLaren, P. (2005). Social relationships as predictors of depression and suicidal ideation in older adults. *Aging and Mental Health*, 9, 517-525.
- Verhagen, M., Van der Meij, A., Franke, B., Vollebergh, W., De Graaf, R., Buitelaar, J., et al. (2008). Familiality of major depressive disorder and gender differences in co-morbidity. *Acta Psychiatrica Scandinavica*, Retrieved February 6, 2008, from <http://www.ncbi.nlm.nih.gov/sites/entrez>.
- Wang, H., Mittleman, M., Leineweber, C., & Orth-Gomer, K. (2006). Depressive symptoms, social isolation, and progression of coronary artery atherosclerosis: The Stockholm Female Coronary Angiography Study. *Psychotherapy Psychosomatics*, 75, 96-102.
- Westin, L., Carlsson, R., Erhardt, L., Cantor-Graae, E., & McNeil, T. (1999). Differences in quality of life in men and women with ischemic heart disease. A prospective controlled study. *Scandinavian Cardiovascular Journal*, 33, 160-165.
- Westlake, C., Dracup, K., Fonarow, G., & Hamilton, M. (2005). Depression in patients with heart failure. *Journal of Cardiac Failure*, 11, 30-35.
- Williamson, D., Birmaher, B., Axelson, D., Ryan, N., & Dahl, R. (2004). First episode of depression in children at low and high familial risk for depression. *Journal of the American Academy of Adolescent Psychiatry*, 43, 291-297.
- Xie, B., Chou, C., Spruijt-Metz, D., Liu, C., Xia, J., Gong, J., et al. (2005). Effects of perceived peer isolation and social support availability on the relationship between body mass index and depressive symptoms. *International Journal of Obesity*, 29, 1137-1143.
- Zinn-Souza, L., Nagai, R., Teixeira, L., Latorre, M., Roberts, R., Cooper, S., et al. (2008). Factors associated with depression symptoms in high school students in Sao Paulo, Brazil. *Rev Saude Publica*, 42, 34-40.

## APPENDICES

## APPENDIX A

## Patient Health Questionnaire – PHQ-9

Name: \_\_\_\_\_ Date of Birth: \_\_\_\_ / \_\_\_\_ / \_\_\_\_

Age: \_\_\_\_\_ Today's Date: \_\_\_\_\_ Gender: ☐ Male ☐ Female

Race: ☐ Caucasian ☐ Hispanic ☐ African American ☐ Native American

Do you have a past Cardiac History? ☐ Yes ☐ No

Family Cardiac History? ☐ Yes ☐ No

Marital Status: ☐ Single ☐ Married ☐ Divorced ☐ Widowed

A. Over the last 2 weeks, how often have you been bothered by any of the following problems? (mark an X in the appropriate box)

	Not at all	Several days	More than half the days	Nearly every day
1. Little interest or pleasure in doing things				
2. Feeling down, depressed or hopeless				
3. Trouble falling/staying asleep, sleeping too much				
4. Feeling tired or having little energy				
5. Poor appetite or overeating				

	<b>Not at all</b>	<b>Several days</b>	<b>More than half the days</b>	<b>Nearly every day</b>
<b>6. Feeling bad about yourself – or that you are a failure or have let yourself or your family down</b>				
<b>7. Trouble concentrating on things, such as reading the newspaper or watching television</b>				
<b>8. Moving or speaking so slowly that other people could have noticed. Or the opposite – being so fidgety or restless that you have been moving around a lot more than usual</b>				
<b>9. Thought that you would be better off dead or of hurting yourself in some way</b>				



B. If you have been bothered by any of the 9 problems listed above, please answer the following:

How difficult have these problems made it for you to do your work, take care of things at home, or get along with other people? (mark an X under the correct level)

Not difficult at all	Somewhat difficult	Very difficult	Extremely difficult

## APPENDIX B

**INFORMED CONSENT**

January 31,2009

**Detection of Depression in the Hospitalized Cardiac Patient**

**Purpose of the Study:** Martine Geddes, a masters student in the Department of Health, Physical Education and Recreation at Utah State University, under the direction of Dr.Philip J.Waite, is conducting a research study to find out more about depression in the patient hospitalized for an acute coronary syndrome. You have been asked to take part in an effort to better understand if depression is readily detected during a patients stay in the hospital. The results of this study will impact future comprehensive care given to the cardiac patient.

**Procedures/Time to Participate:** If you agree to be in this research study, you will be asked to complete a brief questionnaire that should take approximately 5-10 minutes to complete. When completed, you will give the questionnaire to your nurse, or Martine Geddes, who will then add your questionnaire to the research file.

**Risks:** There is minimal anticipated risks involved in participating in this study.

**Benefits:** There may or may not be any direct benefit to you for participating in this study. However, the knowledge gained from your participation may affect further treatment of depression in the hospitalized cardiac patient as we now know that depression is becoming an independent risk factor for heart disease.

**Explanation and Offer to Answer Questions:** You may ask questions concerning this research and have those questions answered before agreeing to participate. If you have any questions about your rights as a participant in this study that have not been answered by the investigator, you may contact the Institutional Review Board at Utah State University (435) 797-1821 or Dr. Philip J. Waite at (435) 797-1000.

**Confidentiality:** The questionnaires completed for this study will identify you by name, only long enough to code your name and responses onto a specific code sheet. This information will only be viewed by Dr. Phillip J. Waite and Martine Geddes. Your Doctor will have no knowledge of your responses. Research records will be kept confidential, consistent with federal and state regulations. Records for this study will be kept in a locked cabinet in the university investigators office with no specific individual or department identified by name.

**Freedom to Withdraw:** You are free to decide not to participate in this study or to withdraw at any time without adversely affecting your relationship with the investigators or with this institution.

**IRB Approval Statement:** The Institutional Review Board for the protection of human participants at Utah State University has approved this research study. If you have any questions or concerns about your rights, you may contact the IRB Administrator at (435) 797-0567. If you have a concern or complaint about the research and you would like to contact someone other than the research team, you may contact the IRB Administrator to obtain information or to offer input.

**Copy of Consent:** You have been given two copies of this Informed Consent. Please sign both copies and retain one copy for your files.

I certify that the research study has been explained to the individual by me, or by my research staff, and that the individual understands the nature and purpose, the possible risks and benefits associated with taking part in the research study. Any questions that have been raised, have been answered.

/

---

 Signature of Principal Investigator

Dr. Philip J. Waite

Principal Investigator (435) 797-1000

Signature of Student Researcher or Co-PI

Martine S. Geddes

Student Researcher (435) 716-5323

**By signing below, I agree to participate**

---

 Participant signature

---

 Date

## APPENDIX C



## APPENDIX D

Dear Physician,

My name is Martine Geddes and I work for Logan Regional Hospital as the Clinical Lead in the Cardiac Rehabilitation program. I am working on my master's degree in Health Education at Utah State University. As the final fulfillment of my degree, I am working on a thesis and would like to conduct a study in the Intensive Care Unit at Logan Regional Hospital. My topic is "Detection of depression in the hospitalized cardiac patient".

I would like to get your permission to administer a simple, 9 question depression inventory to your patients that come into this unit with either a troponin blood test ordered, hx of CABG, AVR, MVR, or PTCA/Stent placement, ACS, R/O MI, atrial fibrillation, CHF, or unstable angina. The depression inventory I've chosen to use is the Patient Health Questionnaire 9. I have enclosed a copy for you to review. In order to administer this questionnaire, patient informed consent will be obtained, and this is also enclosed for your review. I expect the questionnaire to take approximately 5 minutes to complete and administration will not begin until the patient has reached a stable point in his/her hospitalization. Patients excluded from the study would be those who are demented, on ventilatory support, on hospice, or expire during their hospitalization.

Please review the physician consent form and sign and fax back to me if you are willing to allow me to administer this brief questionnaire to your patients (435) 716-2929. You can also call me at (435) 716-5323.

Your support would be greatly appreciated!

Sincerely,

Martine S. Geddes, B.S.



## Physician Permission Form

I agree to allow Martine Geddes, or a trained data collector working on her behalf, to administer the Patient Health Questionnaire (PHQ-9) to any of my patients that are admitted to the Intensive Care Unit at Logan Regional Hospital between October 2008 and November 2009 with either a troponin blood test ordered, hx of CABG, AVR, MVR, or a PTCA/Stent procedure, ACS, R/O MI, atrial fibrillation, CHF, or unstable angina. I understand that this questionnaire will not be administered until the patient is at a stable point in their hospitalization and informed consent has been obtained.

---

**Physician Signature**

**Date**

Fax to: (435) 716-2929

## APPENDIX E



**Intermountain  
Healthcare  
Office of Research**

8th Avenue and C Street  
Salt Lake City, Utah 84143  
801.408.1991  
800.321.2107  
801.408.6783 Fax  
officeofresearch@imail.org

June 1, 2009

Martine Geddes, B.S.  
2920 Naomi Dr.  
North Logan, UT 84341

**RE:** IRB # 1006263 - Depression Detection  
**Protocol Title:** *Depression Detection in the Hospitalized Cardiac Patient*  
**Meeting Date:** 6/4/2008 (to be reported to the Committee)  
**On Agenda For:** Continuing Review of Research – Expedited Review  
**Approved:** 5/28/2009  
**Expiration Date:** 5/28/2010

Dear Martine Geddes, B.S.:

The above referenced project has been reviewed by a member of the Intermountain Healthcare Urban North Region Institutional Review Board and continuing approval was recommended.

The FDA requires that research projects be reviewed annually, or more often at the discretion of the IRB. You will be notified when it is time for renewal of this study. It is your responsibility to respond to this notification. If you do not respond, approval of this study will be terminated. In the meantime, if there are any administrative, procedural or clinical changes you will need to submit them to the IRB for approval prior to making them effective.

It is your responsibility to notify DHHS and/or the FDA, and the Chairperson of the IRB Committee of any occurrence or emergency that seriously increases the risk to or affects the welfare of subjects.

If you have questions regarding this decision, please contact Mark Hall in the IRB Office at (801) 408-6782.

**Approved Items:**

- Continuing Review Application dated 2/10/2009
- Study Protocol
- Patient Health Questionnaire
- Informed Consent Document

Sincerely,

Richard S. White, MD  
Chairperson  
Urban North Region  
Institutional Review Board

